VOLUME 13 NUMBER 1

ARCHIVES
OF
DERMATOLOGY AND SYPHILOLOGY

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JANUARY, 1926

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $6.00

Entered as Second-Class Matter, October 16, 1917, at the Postoffice at Chicago, Illinois, Under Act of Congress of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 22, 1918.

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Archives of Dermatology and Syphilology

VOLUME 13 JANUARY, 1926 NUMBER 1

DERMATOLOGIC QUACKERY *

HOWARD FOX, M.D.
NEW YORK

"The first and worst of all frauds is to cheat one's self."

The extraordinary prevalence of medical quackery in this somewhat enlightened age, seems of sufficient importance to be chosen as the subject of my address. That medical charlatanry has existed from the earliest times and will not entirely disappear until the millenium arrives is certain and is plainly shown by a perusal of the splendid book of Dr. James J. Walsh,[1] entitled "Cures, the Story of the Cures that Fail." This book, with its historical account of the various medical frauds which have been perpetrated on gullible humanity during many centuries should prove of absorbing interest to every physician. Written, as it is, in the author's delightfully humorous style and without a trace of spite or ill will, it is the kind of book that is most likely to influence the average layman. An interesting little book on the same subject is one which was written anonymously in 1718 by an English physician [2] and entitled "The Modern Quack." The conditions described sound strangely like those of the present day, even if the language is somewhat different with its frequent references to "physic" and "distempers."

PREVALENCE OF QUACKERY TODAY

"More varieties of irregular practice are in existence today than at any earlier period in the world's history," is stated by Lewellys F. Barker [3] in a scholarly address on "The Irregular Practice of Medicine." He adds that "almost all of the inferior methods of practice earlier in vogue, are still more or less prevalent and to them has been added a host of new vagaries of the present." It is a little hard on our proud twentieth century civilization to hear from Walsh that "we are just as susceptible of being influenced by healers and healing methods, absurd in themselves, as any generation of mankind ever was."

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* President's Address at the Annual Meeting of the American Dermatological Association, Washington, D. C., May, 1925.

1. Walsh, J. J.: Cures, the Story of the Cures That Fail, New York, D. Appleton and Company, 1923.

2. The Modern Quack, London, J. Roberts, 1718.

3. Barker, L. F.: The Irregular Practice of Medicine, Oct. 13, 1921.

There can be no doubt about the prevalence of quackery in the United States. This was shown some years ago in the astonishing revelations of Samuel Hopkins Adams,[4] published in Colliers Weekly under the eminently proper title of the "Great American Fraud." The crusade of the *Chicago Tribune* was also a notable one in exposing some of the worst methods of quacks in that city. If any one doubts the present existence of quackery of every conceivable type, let him read the two volumes of Nostrums and Quackery,[5] published by the American Medical Association from articles that have been written by Dr. Arthur J. Cramp, chief of the valuable and fearless Propaganda Department, or as it is now called, Bureau of Investigation.

It is only necessary to glance at the newspapers or street car advertisements to get an idea of the number of nostrums now on the market. It is rather astounding to hear of their increase in recent years. As Dr. S. Dana Hubbard of the New York City Department of Health says, "We are informed that in 1888 there were less than a thousand 'proprietaries.' In 1924, they numbered from a careful estimate of a prominent drug house about 60,000." My informant also states that the patent medicine industries account for about one twelfth of the total advertising of the country. According to a recent article in the Druggists Circular[6] (a periodical representing the best traditions of the drug industry), it was estimated that the annual sales of "proprietary medicines" by drug stores amounted to $275,000,000, more than twice as much as that paid for prescriptions.

SOME CAUSES OF QUACKERY

So much has been written about quackery that the titles alone of the various communications cover nearly fourteen pages of the index catalog of the Surgeon General's Library. Many indulge in speculation regarding its causation. The opinion of Barker is that the essential cause is an economic one, namely, that "it is comparatively easy to make money by quackery." This is true, but why should the public allow itself to be so easily fooled? Ignorance regarding matters of health accounts for much of the evil, but by no means for all of it. Many

4. Adams, S. H.: The Great American Fraud (collected articles on the nostrum evil and quackery reprinted from Colliers Magazine), Chicago, American Medical Association Press, 1912.

5. Nostrums and Quackery (articles on the nostrum evil and quackery, reprinted with additions and modifications, from The Journal of the American Medical Association), Ed. 2, Chicago, American Medical Association Press, 1912. A very instructive and interesting little pamphlet is published by the same press entitled "The Nostrum and Public Health and the Truth in Advertising Products."

6. Drug Store Sales Figures, Druggists Circular **68**:219 (June) 1924.

highly educated people with a reasonable knowledge of the subject of health are often dupes of the most patent forms of medical swindles. The oft quoted paragraph of Adams describes a situation which is hard to explain. "Our national quality of commercial shrewdness fails us when we go into the open market to purchase relief from suffering." One reason for patronizing the quack or using the nostrum is the fact that a sick person or one who thinks he is sick loses, for the time, his good judgment and perhaps his sense of humor. As Walsh says, "most of us have not too much reasoning power anyhow and when we have anything the matter with us, most of whatever little we have is likely to vanish."

Special reasons have been suggested to account for the prevalence of quackery in the United States. It is claimed to be partly due to our democratic form of government, one writer saying it is not an accident that America is at once "the home of quackery and the home of the free." This might seem to be borne out from the prevalence of quackery in England, which is a democracy like ours in all but name. This argument is weakened, however, from the fact that in its autocratic days Germany was infested with quacks, and this in spite of her high standard of medical education. According to Flexner,[7] the number of registered quacks in Berlin increased by 1,600 per cent from 1879 to 1903, although the population increased only 60 per cent. One plausible cause for the spread of quackery in recent years is that we are living in an age of wonderful inventions, when anything, especially in the nature of chemistry or electricity, seems possible.

The shortcomings of the medical profession itself have often been considered as contributary causes at least to the prevalence of quackery. Thus we have been accused of too much drugging and too ready recourse to surgery, conditions which at present have been largely remedied. The mendaciousness of physicians is another charge, the physician being expected, according to Williams,[8] to conceal the truth, with the result that all his statements are habitually received with qualifications. This, I think, is by no means true at the present time, as the average intelligent patient is now most insistent on knowing all about his affliction, especially its causation. Too little attention is paid to the more trifling "ailments" of mankind and the temporary alleviation of subjective symptoms. Where the scientific physician fails in studying the psychic needs of his patient, the quack is at his best. His chief stock in trade is his thorough knowledge of human nature. The honesty of a real physician is often a handicap with certain types of patients. The lack of interest in therapy and the tendency of many physicians to consider writing on the subject as beneath their dignity, is unfortunate. This Association at

7. Flexner, Abraham: Chapter on Sects and Quacks in Medical Education in Europe, Carnegie Foundation Bull. 6, New York, 1912.

8. Williams, T. A.: Quackery and Medical Education, Am. J. Clin. Med. **18**:748 (July) 1911.

least is to be congratulated on its enlightened stand in having lately chosen therapeutics as the main topic for general discussion. The profession is unquestionably at fault in its failure to take an active interest in legislative matters pertaining to health, and in not supporting to its fullest power the activities of its local and national medical organizations.

That quackery should be particularly rampant in this age of popular education might appear to be hard to explain. As a matter of fact, the dissemination of information by the enormous number of newspapers and magazines of the present day, has offered the advertising quack the opportunity he needs. The sad truth is that we have not as yet succeeded in counteracting his harmful efforts by sufficient propaganda.

Whatever the underlying causes of quackery may be, there is no doubt of the technic by which its message is carried to an uneducated and gullible public. It is advertising, enshrouded in secrecy and backed by the ubiquitous testimonial. "According to the estimate of a prominent advertising firm," writes Adams, "90 per cent. of the earning capacity of the prominent nostrums is represented by their advertising." The element of secrecy allows the patent medicine manufacturer to cover up something which may be injurious, dangerous or merely worthless, and gives him the opportunity to make claims for his production which would otherwise be laughed at. As to "testimonials," it is enough to say that many of them continue to be published long after the patient has succumbed to his disease. A rather gruesome joke, which is shown in one of the Educational Posters of the American Medical Association, was the simultaneous publication in a certain New England paper of a "testimonial" and the death notice of the victim.

With the enormous amount of advertising that is carried on at the present time for the sale of ordinary merchandise, we are not concerned. When it has an injurious effect on the public health, however, it is time for us to be heard. "Medicine is not a trade," writes W. J. Robinson,[9] "like selling shoes. If you buy a bad pair of shoes you only lose your money and stop trading with that store. If you lose a child from diphtheria through a quack it is poor consolation to say that you won't call him next time." The question is well stated by Cramp:

> It may or may not be a bad thing for the community, economically speaking, if the public is led through skillful advertising to buy more hats, more pianos or more automobiles than it can really afford or has any legitimate use for, but it requires no argument to prove that advertising which makes a well man think he is sick and a sick man think he is *very sick* with the object of making these men drug themselves unnecessarily is a crime against the public health.

THE RELATIONSHIP OF THE PRESS TO QUACKERY

That advertising is the basis for the very existence of the greater part of quackery is admittedly true. It is equally true that the most favorable medium for such advertising is found in the newspapers and the

9. Robinson, W. J.: Scientific Medicine Versus Quackery, Am. J. Clin. Med. **18**:157 (Feb.) 1911.

magazines. The rôle of the press in the promotion of quackery was ably shown by Champe Andrews,[10] in an address entitled "A Century's Criminal Alliance Between Quacks and Some Newspapers." The subject was also handled without gloves in a most illuminating manner by Adams and by Mark Sullivan. Fortunately, the situation has improved of late years, and at present many of the great daily newspapers and many of the better magazines are almost free from medical advertising, especially of the baser sort. I have been agreeably surprised at the number of high class magazines whose advertising pages are as free from objectionable features as their editorial pages.

In answer to the question "Do you think that the newspapers and magazines have less of a 'criminal alliance' than formerly?" Dr. Cramp wrote me as follows:

> There is no question that the newspapers and magazines are no longer under the control of the quack and nostrum interest as they were fifteen or even ten years ago. The reason is not that the newspapers are more interested in the health or morals of the public, but that the repeated exposures have made the advertising of quacks and nostrums less respectable and therefore the better type of newspapers hesitate to accept it.

The religious and foreign language papers have long been offenders in medical advertising. That many of the former publications have become converted to decent advertising would appear from my recent perusal of seventeen of the more important ones. The majority of these at present are either free from all notices of quackery and nostrums or contain at most a few of the less harmful variety. There are still exceptions that have not as yet seen the light, and which continue their attempts to minister to the spiritual wants of their readers while not disdaining to disseminate injurious information for their bodies and perhaps their souls as well. The only newspaper of prominence which has consistently refused nostrum advertising is the *Christian Science Monitor,* and, as some one has said, it makes up for it by boosting its peculiar brand of quackery in its editorial columns.

Even that hotbed of advertisements of the lowest types of quackery, the foreign language press, has apparently shown some improvement of late. A few, at least, of the thirty-three newspapers published in fifteen languages in New York are relatively free from this stigma. Of two Italian papers, one simply reeks with advertisements of venereal specialists, while the other, whose editorial pages are on a much higher plane, is almost free from medical advertising. As Dr. Nichols[11] of Oregon says, "the foreigner looks upon an advertisement as gospel truth and accepts treatment from an advertisement in preference to honest treatment by a reputable man."

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10. Andrews, Champe S.: A Century's Criminal Alliance Between Quacks and Some Newspapers, New York, Stettiner Bros., 1905.

11. Nichols, H. S.: Discussion of Report on Quackery by H. R. Krasnow, Illinois M. J. **35**:16 (Jan.) 1919.

But what of the advertising in our own medical journals? Seeking information about journals which, through their advertising pages fail to live up to the ideals of the profession, Dr. Cramp has written me as follows:

> They are of varying degrees of badness and I have grouped them in three classes. The first class (nine in number) are journals that in many instances contain original articles from good men and in general conform in their reading pages, at least, to the amenities of decent journalism. The objectionable part of such journals is their advertising pages. The second group (seven in number) are those that not only have advertising pages that are a disgrace, but whose reading pages can hardly be considered scientific or high class. The third group (three in number) are the utterly hopeless ones that apparently exist merely for the purpose of making money for those who run them.

The problem of preventing the quack from swindling the public by means of the radio, must be faced sooner or later. In fact, it has recently been brought to our attention in New York that a certain advertising doctor has disregarded all regulations by repeated exploitation of the public through appeals by radio. Notices of these talks were duly announced in the papers which print such stuff. It is hoped that definite regulations by the Department of Commerce will shortly put a complete curb on this method of swindling the public.

THE FOOD AND DRUGS ACT

A partial protection against medical fraud is furnished by the federal Food and Drugs Act, popularly known as the "Pure Food Law." This requires that any preparation containing one or more of eleven specified drugs or their derivatives shall have their names and amounts printed on the label. The list of these drugs includes alcohol, morphin, opium, cocain, heroin, alpha and beta eucain, chloroform, canabis indica, chloral hydrate and acetanilid. The law also says that statements on the trade packages shall not be "false or misleading" in regard to the composition of the product or "false and fraudulent" regarding its therapeutic claims. The joker in this law is that while misleading and false statements cannot be made on the trade package, the manufacturer or exploiter of the nostrum may lie to his heart's content about it in the newspapers or elsewhere. In addition to federal restrictions, there are also statutes relating to pure food and drugs in various states.

One of the omissions in the federal law is that it does not affect the sale of cosmetics unless they are sold with therapeutic claims. At the request of Dr. Harold N. Cole, a resolution was introduced by me last year in the House of Delegates of the American Medical Association urging the regulation of the sale of cosmetics by compelling the printing on the label of the names of all the poisonous ingredients. The resolution was unanimously passed. A bill was recently introduced in the New Hampshire State legislature designed to prevent the sale of cosmetic preparations containing arsenic, mercury, lead, copper, silver or para-

phenylendiamin. I am informed by Dr. Dennis Sullivan of Concord that the bill "passed the House but was killed in the Senate due to the opposition of the druggists, who claimed that its passage would not prevent the sale by mail order houses in other states, thus driving home business to outsiders." In other words, it would appear to be proper that regulations relating to harmful ingredients of cosmetics should be promulgated by federal rather than by state authority.

MEDICAL CULTS WITH SPECIAL REFERENCE TO CHIROPRACTIC

The forms of quackery have never been more manifold and varied than at the present time. A year ago, the *Weekly Bulletin* [12] of the New York Department of Health requested physicians and other citizens to register complaints relative to the irregular practice of medicine. As indicative of the versatility of quacks, a list of fifty-nine different types was printed, which included such extraordinary names as "biodynamo-chromatic" therapy, "Christos" (blood washers), "electronaprotherapy," "geo-therapy," "leonic healers," "naprapath," "sani-practors," "telathermy" and "vitopaths."

The long list was kind enough to omit osteopathy and chiropractic. As the last mentioned cult is for the time being so much in the public eye, it deserves special attention. There is an interesting and entertaining little pamphlet (price, $1) entitled "Major Adjustments" edited by two professors of chiropractic. It contains an alphabetical list of 1,017 diseases, ailments and conditions with suggested "adjustment centers." "The chiropractor," it says, "will find his adjustments may be depended upon to produce permanent results, if given at the places indicated." Following this is a key of abbreviations placed after each disease such as D. for dorsal, K.P. for "kidney place," G.A. for "general adjustment" or R.C., which occurs rather infrequently, and signifies "remove cause." There are only three conditions to which the words "no cure" are appended, including epithelioma, carcinoma and sarcoma. In spite of carcinoma being listed as a no cure affair, "cancer of the stomach" is not thus tagged and would appear to be curable. No explanation as to how it differs from carcinoma is offered. One is simply told to "remove cause" and incidentally do a little adjustment at L.Z. (lower zone) and K.P.

Our chiropractic friends tackle everything by spinal adjustment, including such simple affairs as smallpox, yellow fever, Asiatic cholera and bubonic plague. Think of the crime of an ignorant man, or for that matter the most intelligent and educated one, treating by these methods such conditions as cerebrospinal meningitis, bronchopneumonia, poliomyelitis and strangulation of the intestines. The field of dermatology is well covered by adjustments, ranging from dandruff and eczema to scleroderma, locomotor ataxia, syphilis and leprosy. Those who have struggled with psoriasis can take heed and adjust at C. 3-5, D. 10-12

12. Weekly Bulletin, Dept. of Health, New York City, **13**, Feb. 16, 1924.

and C.P. (central or upper liver place). To cover the ground completely they treat color blindness, "thrills," dribbling, waddling gait and leukocytosis and one of these hitherto undescribed diseases, "pupillary skin."

DERMATOLOGIC NOSTRUMS AND COSMETICS

Some of the widely advertised nostrums for diseases of the skin are not particularly harmful except as they encourage self medication by those who do not know that their supposed eczema may in reality be tuberculosis or syphilis. "Affections of the skin of entirely different natures, so closely resemble each other," wrote Ticknor [13] in 1839, "that a practiced eye alone can make a discrimination between them. Is it possible then for an ignorant man or weak member of the sisterhood to cure all diseases of the skin with a single favorite nostrum?"

The following is a sample of some of the dermatologic humbug perpetrated on the public by a certain mail order house. Of the fourteen different remedies offered by this particular company, all were found on examination to be essentially the same in composition, differing only in flavoring. The versatility of remedy No. 6 was shown by its recommendation for "eczema, pimples, skin diseases and smallpox." It contained about 25 per cent alcohol, about 50 per cent olive oil and a few drops of oils of spikenard; cloves and anise.

Whatever some may think about the simpler nostrums for skin affections, there can be no difference of opinion regarding the utter depravity of those who prey on the unfortunate victims of cancer. Practically all the nostrums advertised and guaranteed as "sure cures" for cancer of the skin are of the escharotic type, about evenly divided between zinc and arsenic. The effrontery of one of the most notorious of this class of quacks is shown by a statement in his "hurry up" letter. "When I pause," he writes, "and consider the amount of quackery and humbuggery practiced all over the country it is not difficult to understand why the afflicted hesitate to accept new treatment, no matter how logical it may be," The cheek of this particular gentleman is almost equal to that of the humorist who put "humbug oil" on the market.

In a discussion of the subject of cosmetics it is not my function to express an opinion regarding their esthetic value. It is my function and my duty to say a few words regarding some of their harmful effects. If one is too near sighted to appreciate the prevalence of cosmetics at the present time, let him glance over the advertisements in a certain class of our newspapers and magazines. He will be astounded at the number relating to cosmetic nostrums and remedies for the cure of skin diseases. In one of our most sensational illustrated dailies in New York the proportion of such advertising was recently found to be 60 per cent of

13. Ticknor, Caleb: Exposition of Quackery and Imposture in Medicine, London, James S. Hodson, 1839.

the total. According to an official report from the Department of Commerce,[14] the manufacture of perfumery and cosmetics is the only branch of the drug industry which shows an uninterrupted growth from census to census (1849-1921). The estimate of expenditures for these articles (perfumery and cosmetics are always classed together) in 1921, based on tax collections, was $150,000,000. According to the *Druggists Circular,* the American public requires each year 240,000,000 packages of talcum and face powder and nearly 18,000,0000 packages of rouge, among other toilet accessories.

The possible dangers of cosmetics are shown in a study by Wilbert [15] entitled "Cosmetics as Drugs," the author being an expert government pharmacologist. Among the preparations classed as cosmetics he includes hair tonics, hair washes, hair dyes, shampoo powders, complexion powders, toilet powders, freckle lotions, face enamels, toilet waters, toilet vinegars, grease paints, face creams, liquid enamels, beauty washes and skin bleaches, truly a formidable list. The writer discusses the dangers resulting from the use of wood alcohol, paraphenylendiamin and the salts of lead, mercury and bismuth. "To prevent serious intoxication and to preclude obvious deception and fraud," he urges that "cosmetics should be classed as drugs and proprietary preparations sold as cosmetics should be required to state on the label the name of any poisonous ingredient that may be contained therein." The use of wood alcohol in preparations for external use, he very rightly says, should be prohibited by law.

The subject of cosmetics has recently been discussed by Cole [16] as a chairman's address and by Klauder [17] in *Hygeia.* You are all doubtless familiar with the pamphlet on "Cosmetic Nostrums and Allied Preparations," [18] published by the American Medical Association and containing information concerning about 130 preparations. I know of no more profitable way for a dermatologist to invest fifteen cents than to order a copy of this pamphlet.

BEAUTY PARLORS

The modern beauty parlor appears to supply a legitimate demand for an increasing number of patrons. As long as its activities are confined within proper limits we cannot quarrel with it. When, however,

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14. The Drug Industries, Dept. of Commerce Bureau of the Census, Washington, D. C., 1924.

15. Wilbert, M. I.: Cosmetics as Drugs, Pub. Health Rep. **30**:3059 (Oct. 15) 1915.

16. Cole, H. N.: The Dermatoses Due to Cosmetics, J. A. M. A. **82**:1909 (June 14) 1924.

17. Klauder, J. V.: The Skin and Cosmetics, Hygeia **2**:91 (Feb.) 1924.

18. Cosmetic Nostrums and Allied Preparations, Chicago, American Medical Association Press, 1921.

the head of a certain widely advertised beauty parlor, calling herself a "face specialist," states that she "guarantees permanently to remove smallpox pittings and all blemishes of the skin," we can justly claim that she is practicing medicine without a license. The same thing is done in many of the concerns of this class in New York. In the boroughs of Manhattan and the Bronx there are 1,177 hair dressing establishments, many of which are actually beauty parlors which practice medicine illegally. Our health authorities are so occupied, however, with bigger game that the comparatively harmless activities of the beauty parlor have been largely ignored. Another beauty establishment claims to have a preparation that will positively remove superfluous hair, "causing it to grow softer and finer at first, then to disappear altogether."

Aside from their dabbling in medicine, an objectionable side of the beauty parlor is the parting of silly women with their money, which after all does not concern us. A certain establishment with advertising pamphlets that are really a work of art, strongly recommends their "face moulding treatment" for wrinkles. In describing their supposedly novel method, they say, "Here we do not think in terms of cosmetics but of physiology." In the practice of their method they use such high sounding preparations as "rose leaf cleanser" followed by a "skin freshner." The face is then manipulated with "face moulding cream," according to minute directions shown by a "muscle chart." If there are any deep wrinkles, "balsam tissue astringent" is also used, and the performance ends with an application of ordinary plain "balsam astringent," whatever that might be. It is a curious coincidence that the name which this particular beauty parlor bears is that of a plant which in susceptible persons causes a dermatitis as bad as that of poison ivy.

In my casual study of beautifying advertisements I have not seen anything equal in foolishness to the following quotation which was part of an article submitted to me for honest criticism by an advertising firm. I gave them my opinion. The passage in question advised the candidate for beauty as follows:

> Sit before a mirror resting the elbows on a small cushion, so as not to coarsen the skin of the elbows. Dip tips of first three fingers in . . . cream. Apply finger tips to the skin and press gently, first, second, third, until you have counted thirty. The pressure should be *very gentle,* not quite as heavy as that used in *pressing an electric button. Do not strike* as though playing a piano, as this will injure the delicate underlying nerves.

COSMETIC SURGERY

The stigma that attaches to cosmetic or beauty surgery is that it has been so thoroughly exploited by quacks. A reputable surgeon should not be criticized for performing any operation for cosmetic purposes any more than a reputable dermatologist for removing a nevus araneus.

Both are done solely to improve the patient's appearance. As the majority of surgeons have continued to look askance at this work, the insistent demand by a certain class of the public has been willingly supplied by the advertising quack. Some of their results are passably good, while others are decidedly bad.

The unfortunate results obtained by one such man were probably responsible for his entering on a mail order business of removing wrinkles and blemishes at long distance. This gentleman, who hails from a western city, is considered by hundreds of pleased women and men, according to his advertisements, to be "the world's leading dermatologist, plastic surgeon and beauty specialist"—honors enough for any man. His circular goes on to say that "Dr. . . . 's practice is so large in his office here that he has occupied the unique position of having advertised for people to *stay away from* his offices instead of coming to them." Of the many wonders which he claims to perform through his "private, personal and secret processes and preparations," that produced by preparation No. 15 seems to be the last word in cutaneous medicine. This is the . . . facial "uplift" and "instant deep wrinkle and line remover." It claims to be "mechanical in its action" and to cause the complete removal of all wrinkles and lines in five minutes, the treatment having to be repeated only at intervals of about one year.

A method of beauty surgery which has been much employed by quacks is the subcutaneous injection of paraffin. The frightful disfiguration that results at times from this procedure is familiar to every dermatologist. In addition there are cases on record of retinitis, optic neuritis and sudden blindness, which have followed such injections of paraffin. Dr. J. Eastman Sheehan [19] states that "obviously such material is not one to depend upon for permanent satisfaction. Indeed its employment should be interdicted altogether." According to Seymour Oppenheimer,[20] "the paraffin method of cosmetic rhinoplasty is dangerous even in the hands of the well equipped surgeon and doubly more dangerous and to be avoided at the hands of the ignorant, unscrupulous and uneducated beauty doctor." There are evidences that some of the quacks are beginning to be afraid of this method, as shown by a recent pamphlet of a certain "facial lifter" in New York who incidentally uses glandular therapy as an adjunct in his "rejuvenation" work.

An organization was recently formed in London and later transferred to Paris, which may aid in the war on advertising beauty doctors. That,

19. Sheehan, J. Eastman: Plastic Surgery of the Face, New York, Paul Hoeber, Inc., in press.

20. Oppenheimer, Seymour: A Condemnatory Note on the Use of Paraffin in Cosmetic Rhinoplasty, Laryngoscope **30**:595 (Sept.) 1920.

I am told, is one of the purposes of its formation. The organization in question is the International Clinic of Oto-Rhino-Laryngology and Facio-Maxillary Surgery, with headquarters at the St. Louis Hospital in Paris. Its main object is to preserve and spread the knowledge of plastic surgery gained during the war.

VENEREAL DISEASES

The lowest and most degrading form of quack is the man who preys on the victim of venereal disease, unless it is the creature who sells narcotic drugs as "patent medicine" to drug addicts. Fear of disclosure on the part of the venereal patient makes him an easy victim to the blackmailing practices of the quack. Thank heaven in this particular phase of quackery some progress in the right direction has been made! I am informed by Dr. Walter M. Brunet, medical director of the American Social Hygiene Association, that there are twenty states which have statutes forbidding the advertising of venereal disease cures; they are Alabama, California, Colorado, Iowa, Kentucky, Louisiana, Massachusetts, Michigan, Minnesota, Nebraska, New York, Oklahoma, Oregon, Pennsylvania, Texas, Utah, Virginia, Washington, West Virginia and Wisconsin.

There are also seventeen states, according to my informant, which forbid the sale of medicine for the treatment of venereal disease without the prescription of a physician; they are Alabama, Colorado, Florida, Kentucky, Louisiana, Maine, Michigan, Minnesota, Missouri, New Hampshire, New York, Oklahoma, Oregon, South Carolina, Utah, West Virginia and Wisconsin. Twelve states appear in each of these honor rolls.

Just how these laws are actually enforced in each community, I am unable to say. In answer to a request for an opinion on this question, Dr. Harry G. Irvine has written me as follows:

> Generally speaking, I think the work on venereal diseases in most of the states has lapsed somewhat due to lessened interest and lack of the stimulation which was present during the war and in others due to lack of financial support. In some states it is undoubtedly going on very well. In facing decreasing appropriations one has to cut where one can, and this usually means educational work, law enforcement and social service. Laboratory work and medical work cannot easily be cut as the amount of work cannot be controlled.

The letter of the law relating to venereal disease advertising is now carried out reasonably well in New York. Thus we no longer see the crude advertisements speaking of syphilis, gonorrhea and "lost manhood." The spirit of the law, however, is often evaded by the use of such terms as "blood and personal diseases." In much of the foreign language press we still see the familiar "old Dr." advertisements. Some good has undoubtedly been accomplished by the propaganda work of

social organizations. The health exhibit for men held at Coney Island in 1916 was the first of its kind limited to men and offering a medical advisory service of the proper kind. In the course of two months, the exhibit was visited by 19,290 men. Its success should encourage other similar ventures.

THE "VIOLET RAY" FAKE

The enormous amount of publicity given the electric toy known as the "violet ray" apparatus threatens to rival that of Perkins tractors, which flourished more than a century ago. The public has heard a good deal about ultraviolet rays of late years and doubtless associates this with the little high frequency apparatus which does not emit any such rays. The medical profession is well aware of the value of high frequency apparatus used for desiccation and electrocoagulation. It does not, however, consider that the vacuum electrode attachment can do more than furnish a mild form of counterirritation. It is the type of apparatus which is now so widely advertised for the cure of almost all the ills to which flesh is heir.

In itself the violet ray apparatus is harmless, as the manufacturers say. But is it harmless to claim, as one of the advertising pamphlets does, that "in the incipient type of tuberculosis, exceedingly good results can be obtained and tubercle bacilli eliminated by the use of the ozone attachments?" Is it honest to allow the public to attempt self treatment of even "surface cancers not too far developed?" How shamefully misleading it is to recommend it for the treatment of diphtheria.

A certain pamphlet advertising this newest plaything will interest the dermatologist by its claims to "quickly remove pimples, blackheads, boils, warts and other blemishes" and its ability to "remove" (apparently not so quickly) "dandruff, eczema and itchy scalp and skin diseases." Like many other nostrums, it claims to cure gray hair. It gives a list of eighty-four pathologic conditions, modestly admitting that not all cases will be cured through high frequency alone, but that "in every case it is a valuable adjunct to other treatment." It makes at least one statement which I believe is truthful, namely, that "all modern beauty specialists use the violet ray in the treatment of the hair and scalp."

Another booklet extolling the virtues of the violet ray recommends its use for ninety diseases alphabetically arranged from abscess to wrinkles. The list includes alcoholism, drug addiction, Brights disease, cancer, diabetes, diphtheria and about twenty-six dermatologic conditions, including poison ivy dermatitis. The average ophthalmologist must gasp to learn that this same toy is recommended for conjunctivitis, trachoma, iritis, incipient cataract, retinitis, atrophy of the optic nerve, paralysis of ocular muscles, intra-ocular hemorrhage and glaucoma.

ROENTGEN-RAY QUACKERY AND HYPERTRICHOSIS

While the "violet ray" apparatus may be an essentially harmless plaything, unless it is used for diphtheria or similar ills, the same cannot be said of the roentgen ray. In the hands of the experienced operators who use all proper safeguards, the roentgen ray has proved of the greatest value in the treatment of skin diseases. It is only after many trials and tribulations that a practically safe technic has been devised for its use which has gained the confidence of the profession and the public to a considerable degree as well. We are now threatened with a condition which if not remedied may seriously affect the use of one of the most essential aids to the modern practice of medicine, the roentgen ray. The menace to which I refer is the recent adoption of this agent by quacks for the treatment of superfluous hair. In their unscrupulous advertising, they quote the texts of Pusey, Jackson, MacKee, Schamberg and others to the effect that the roentgen ray causes the permanent fall of hair. This is perfectly true, but they neglect to say that these authors condemn its use for this purpose on account of the danger of seriously injuring the skin. They do not tell the public that such is the consensus of opinion of all experienced roentgenologists.

An insidious danger in this new form of quackery is that while unquestionably using the roentgen ray, two of the most ambitious of these charlatans either state directly or infer that they do not use the roentgen ray but some mysterious new "wave" or "wave length." During a recent meeting at the New York City Department of Health one of these gentlemen appeared by request, when he was forced to admit, after considerable questioning, that his "invention" was a roentgen-ray tube.

Another of the most persistent advertisers of this class was formerly a reputable member of the profession, connected at different times with several medical schools in New York City. Of this particular renegade the advertising pamphlet speaks in the following glowing terms: "The name of this scientist who attempted the solution of the seemingly impossible is now famous around the world. The most eminent men of his honored profession speak his name with homage and respect and scholarly assemblages listen with dignified approval whenever he makes an address upon the subject that is his recognized specialty." He not only conducts a thriving institution in New York City, but rents his machines for use in different parts of the country, I am informed, for amounts varying from $2,000 to $10,000. In addition, a certain percentage of the lessee's profits are to be turned in at frequent intervals.

At the present time there are four advertising institutions in New York to which women are flocking in great numbers for the treatment of superfluous hair by the roentgen ray. Three of these were personally visited by my office assistants. The technic of treatment varied con-

siderably, as did the fees, the latter ranging from $400 to $1,200 for a complete course of treatment of an extensive case. One of these advertising gentlemen was extremely frank about his work, saying that he used "ordinary x-ray," and that he followed MacKee's textbook. Even if the roentgen ray were a safe and proper method for hypertrichosis, this particular gentleman is certainly heading for trouble. When that arrives we will find him following the usual custom of doing business in another state under a new name. Observing the technic, my informant found that the tube distance was simply measured by the operator's eye. Being told that a current of 2 milliamperes was used, my informant suggested to the operator that the meter was not accurate, receiving the reply, "Well, it's between two and four anyway." It was also interesting to learn that in two of these institutions the names of two of the largest manufacturers of roentgen-ray apparatus were found on the cabinets (supposedly containing the transformers).

The best proof after all that these particularly dangerous quacks are really using the roentgen ray is seen from some of the examples of radiodermatitis which are making their appearance. Nothing but radium or the roentgen ray would produce these characteristic changes. One of the remedies for this evil is for every physician who sees these unfortunate results to write the full particulars to the Bureau of Investigation of the American Medical Association which is collecting data on this subject.

REMEDIES FOR QUACKERY

While it is generally agreed that quackery can never be entirely eradicated, it can be largely prevented, as Barker says, by making it "unprofitable, disreputable and penalizable." This can be accomplished only by the slow and difficult process of educating the public in regard to matters of health. Legislation can then be enacted, and, what is more important, enforced. The greatest difficulty in the education of the public is the fact that much of the press is still allied with the "patent medicine" interests. Every possible encouragement should be given the newspapers and magazines which have shown the courage and decency to rid themselves of patent medicine and quack advertising.

Valuable propaganda can be carried on by influencing the heads of industrial organizations, life insurance companies, etc., and possibly by occasional lectures in high schools and colleges. Much useful knowledge has undoubtedly been disseminated through the societies for the control of cancer, prevention of tuberculosis, social hygiene, etc. The enormous amount of valuable work done by the various councils, and especially by the Bureau of Investigation of the American Medical Association, is generally appreciated by the profession. One of the most promising lines of attack is the appeal to the self-interest of the advertising asso-

ciations showing them that the fraudulent claims of nostrum venders and quacks tend to destroy public confidence in all advertising.

A most effective argument in discussing quackery with laymen is to explain the shameful betrayal of confidence of all who carry on a correspondence with patent medicine concerns or advertising doctors. If you tell the layman that the quack's medicine or treatment is likely to do harm, he may or may not believe you. Tell him rather what happens when he communicates by letter with such men whose letter heads are addressed "dear friend" and who invariably state that all communications are "strictly confidential." As soon as the victim's money is exhausted, his letter is for sale through letter brokers, to brother quacks who desire a mailing list of "easy marks." The confidential information is then public property for the world to read. Adams spoke of a certain warehouse in New York which had seven million such confidential letters for sale.

In order to remedy some of the minor causes which are thought to contribute to the practice of quackery, the physician should pay more attention to the simpler "ailments" of mankind, should study the psychic needs of his patient and take a more serious interest in therapy. He should not consider the discussion or publication of therapeutic papers as beneath his dignity. He should certainly take a more active interest in legislation pertaining to public health than does the average member of the profession, and should aid in the suppression of medical journals with objectionable advertising by at least refusing to contribute to their editorial pages.

Every self respecting physician should be a member of his county medical society, even though he has not the necessary time or inclination to attend the meetings. It then requires only a slight additional effort and cost to become a member of our great national association which is doing so much to carry on the fight for the protection of the public health as well as for the education of the profession. The physician will increase the respect for his profession if he himself realizes and makes the public realize that the eternal struggle against quackery is not a selfish effort on his own part, but is carried on for the protection of the health of all his fellow men.

"KERATODERMIE BLENNORRHAGIQUE" (VIDAL)

REPORT OF A CASE

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The clinical syndrome known as "keratodermie blennorrhagique" was so named by Vidal (1893), who concluded after the observation of one such case, which he recorded in detail, that the condition should be regarded as a distinct clinical entity. His original description specified a generalized and symmetrical cutaneous eruption consisting of cornified crusts with disintegration and destruction of the finger and toe nails, "associated with urethritis and polyarthritis," due to systemic invasion of Neisserian diplococci. His patient also exhibited conjunctivitis and blepharitis of presumably identical origin. The existing cutaneous phenomena, which were described in minute detail, consisted of small, hard, dry cornified crusts "distributed over the head, face and trunk." On the extremities, especially the hands and feet, the lesions were larger and more abundant, the palms and soles presenting numerous yellowish-brown, symmetrical discrete and confluent crusty and cornified areas. On the distal phalanges of the fingers and the toes were thickened, cornified lesions with elevation of the nails. Some of the smaller lesions were described as "semi-transparent, resembling droplets of yellow wax." Removal of individual crusts disclosed verrucous, dry or slightly moist papules. Only exceptionally was it observed that vesiculation or pustulation preceded cornification. Microscopically the cornified areas showed stratifications "resembling a cutaneous horn." Bacteriologic examination of material from the surface lesions was negative. Because of the rupial character of the papular lesions and the general adenopathy present, antisyphilitic medication was instituted by Vidal, but neither the course of the eruptive phenomena nor the lesions then existing were influenced in the least thereby. "With the subsidence of the blennorrhea and arthritis involution of the cutaneous manifestations occurred."

Although Vidal's case record appeared in 1893, not until nearly twenty years later (1912) was a similar observation (Simpson) published in American medical literature. The second case in this country (Roark) was reported in 1916. So far as disclosed by diligent search of available indexes, less than seventy authentic examples of "keratodermie blennorrhagique" have thus far been recorded in the literature of the world. It is, of course, likely that many cases remained undiagnosed or unrecognized.

The most interesting and valuable review of the subject that I have seen is by Keim,[1] who reports one personally observed case with carefully compiled anatomic, histopathologic and other findings accompanied by excellent illustrations. Some of the data incorporated herein are abstracted from Keim's paper.

Since the publication of Vidal's case report, it has been considered that Neisserian diplococci are directly and solely responsible for production of the symptomatologic triad ("urethritis, polyarthritis and hyperkeratosis") which he so graphically described. However, so far as the cutaneous eruptive phenomena are concerned, the etiologic relationship does not appear to have been definitely proved, although, contrary to opinions hitherto prevailing, it is now well understood that Neisserian organisms may invade every structure, fluid and tissue of the human economy. Moreover, so-called "metastatic gonorrhea" implicating various body tissues, also so-called "gonorrheal septicemia," are sufficiently common to demand consideration. However, that the organisms of Neisser possess an elective or selective affinity for certain organs or tissues, as has been claimed by some observers, is merely a theoretical assumption which lacks adequate confirmation.

Granting the doubtful premise that systemic invasion of Neisserian organisms may be solely responsible for the production of so-called "keratodermie blennorrhagique," how can one explain why the observed and presumably characteristic cutaneous manifestations occur in association with "urethritis and polyarthritis" in one instance and not in ten thousand others under identical circumstances and conditions? Moreover, why are females almost invariably exempt from the abnormal occurrence? So far as revealed by literary research, only one case (Isaac) has been recorded in a female. It is known, also, that children are not exempt from invasion of Neisserian organisms, yet no authentic case of so-called "keratodermie blennorrhagique" has ever been reported in a child. Another astonishing fact is that in only a single instance (Wadsack) have Neisserian diplococci been positively isolated from the cutaneous lesions of "true keratodermie blennorrhagique," and rarely have they been recovered from the blood stream.

Based on existing information, therefore, the most that can be said is that the identity of the essential etiologic factor is uncertain. The triad of symptoms "urethritis, polyarthritis and hyperkeratosis" may have a common origin which may be the diplococcus of Neisser, but the relationship thus far remains unproved.

While in Vidal's classical description there is included a "generalized and symmetrical cutaneous eruption," in the majority of cases subse-

1. Keim, H. L.: Arch. Dermat. & Syph. **9**:423 (April) 1924.

quently reported the lesions have been largely confined to localized areas, generally the hands and feet, including finger and toe nails, sometimes the scalp and other portions of the body surface. In the case herein recorded there were a few isolated lesions on the scalp and elsewhere, but the hands and feet constituted the principal sites of the eruptive phenomena. This is in accord with the observations of others who have recently encountered similar cases, and in no sense nullifies or modifies the description given by Vidal.

During the last few years, since the publication of the article by Adamson (1920), there has been considerable discussion concerning the relationship between or the identity of psoriasis and "keratodermie blennorrhagique." It would appear that careful histologic investigation and clinical study should dispel doubtful impressions and clarify prevailing confusion, since there are several essential differences in the mode of development and progress of the two conditions. Psoriasis does not originate in vesicle formation, then proceed to pustulation and cornification; whereas these three successive stages are almost invariable characteristics of "keratodermie blennorrhagique;" moreover, there are relatively slight resemblances clinically between the two disorders. It is, therefore, my conviction that they are not related or identical, but represent two essentially different clinical entities.

From a histopathologic standpoint, the progress and development of the cutaneous manifestations in "keratodermie blennorrhagique" may be conveniently divided into three stages, the minute changes (excerpted from Keim) occurring in the following sequence:

1. The Stage of Vesiculation: The earliest lesion begins in the upper portion of the prickle-cell layer. The vesicle is rounded and has a distinct roof of one or two layers of prickle cells, overlying which is a thinned stratum corneum. The base of the vesicle is composed of moderately acanthotic epithelium. The contents consist for the most part of serum with a few polymorphonuclear and mononuclear leukocytes. Below and immediately surrounding the vesicle in the corium is a distinct infiltration composed mostly of mononuclear lymphocytes with a few polymorphonuclear neutrophils and plasma cells.

2. The Stage of Pustulation: The next stage in development is the rapid evolution of pustulation. The vesicular roof shows signs of disintegration, and the lesion is filled with polymorphonuclear and mononuclear cells, together with a small amount of fibrin. The rete pegs immediately below the lesion are thickened and invaded by small round cell leukocytes and a few plasma cells, and the reaction in the cutis is more marked, extending well upward into the interpapillary tufts. The contents of the pustule, which are about to break through the overlying thinned stratum corneum, show beginning disintegration with increase in pustular elements, fibrin and cells, leading to crusting.

3. The Stage of Crusting. In this stage there is beginning formation of a thick crust, due to drying of the purulent contents, together with considerable parakeratotic epithelium. Confluence of adjacent lesions has occurred, with marked increase in cuticular reaction and edema of the interpapillary tufts. The corium presents a definite mononuclear cellular infiltrate about the follicles, glands and vessels, with decided thickening of the vessel walls. Older lesions show marked flattening of the horny covering with beginning separation. The thickened and elongated rete pegs and collagen increase are striking features of the late crusting stage. The last stage is represented by slight atrophy in the larger lesions resulting from healing below the rupial crust which is then thrown off.

When one remembers the constant and practically invariable association and sequence of the triad of symptoms "urethritis, polyarthritis and hyperkeratosis," it would appear that there should be no diagnostic confusion. However, owing to the comparative infrequency of cutaneous manifestations in association with Neisserian urethrorrhea and arthritis, and the fact that prompt disappearance of the cutaneous lesions is the rule when the infective focus has been eliminated, doubtless in many instances the relationship has been entirely overlooked or has failed of recognition. So far as my knowledge extends, the syndrome described occurs only in the presence of Neisserian germ invasion, i. e., no other type of urethral infection has been known to eventuate in manifestations identical in development, progress and sequence. The progression of the cutaneous lesions from vesiculation to pustulation and finally to cornification may be considered fairly pathognomonic; and when preceded by Neisserian urethrorrhea and polyarthritis, there should be little diagnostic uncertainty.

The local treatment of the cutaneous lesions is for the most part unavailing, for obvious reasons. Since both the articular and cutaneous lesions are metastatic phenomena, apparently the result of systemic invasion of Neisserian diplococci from the original infective focus in the urethra, therapeutic measures must be directed toward elimination of the primary focus. This sounds simple enough, but is not always easy of accomplishment. Metastatic lesions do not develop until the original disease has progressed to varying stages of chronicity, which means extension of the process to the posterior urethra, prostate gland and seminal vesicles, from which sites elimination of infective foci is sometimes a difficult undertaking.

Various therapeutic procedures are indicated to combat the local and general symptoms. The local treatment should be virtually the same as for posterior urethritis, prostatitis and seminal vesiculitis without articular or cutaneous complications, i. e., applications and instillations

of silver nitrate solution or the milder silver salts, as may seem indicated, massage of the prostate and seminal vesicles, the application of heat, etc. General measures include the administration of vaccines and the internal or intravenous introduction of certain drugs to overcome the systemic infection. Obviously no improvement can be expected in the articular and cutaneous complications until the original focus of infection has been eradicated.

In the case herein presented the greatest benefit seems to have accrued from intravenous injections of mercurochrome in 1 per cent. solution. Of course other antigonorrheal medication was also vigorously applied. Applications of heat (electric) to the hyperkeratotic areas, hot saline compresses, and the local use of mercurochrome proved of benefit.

REPORT OF A CASE [2]

History.—S. M., a white man, aged 27, a fireman, admitted to the Louisville City Hospital, Jan. 10, 1925, complained of a severe burning sensation in the urethral interior during micturition, pollakiuria, slight dysuria and painful swelling of the left ankle. The family and previous personal history were of no especial importance. The patient had parotitis and pertussis in childhood; he had had no other illness. He had been married six years and was the father of four living children. He said that his wife had had no miscarriages.

The patient said that about four months prior to entering the hospital, "he passed a worm-like mass" from his urethra while urinating, and there immediately followed a slimy, bloody discharge. Pollakiuria had gradually increased and was always accompanied by a burning sensation and pain, beginning at the distal penile extremity and progressing backward toward the rectum. Two weeks after onset of the trouble he noticed a yellowish, apparently purulent, discharge from the meatus, but he said that "he took some medicine which stopped this."

Two months before admission the patient first noticed some dark, slightly painful areas beneath the nails of the middle and little fingers of the left hand. These spots continued to enlarge; the distal extremities of the nails became elevated from pressure of the underlying structures, and the lesions gradually increased in area until both nails disintegrated and were destroyed. The condition soon extended to the other finger nails, and shortly thereafter the toe nails became similarly involved.

Soon after admission to the hospital there was marked increase in pain and swelling of the left ankle. Roentgen-ray examination showed slight changes in the articular surfaces of the navicular and os calcis, the medial malleolus and astragalus and the cuneiform bones, suggesting a mild infectious arthritis. The right ankle, both knees, elbows and wrists became successively implicated.

Pollakiuria continued to increase (diurnal ten, nocturnal twelve to fourteen) and the patient still complained of pain and burning on micturition. Difficulty was sometimes experienced in "keeping the flow going." The urine was cloudy in appearance; no calculi nor granules were voided at any time; there was an occasional sharp lumbar pain during urination. Urethral discharge was present but not profuse.

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2. This case was briefly described, and the patient was exhibited before the Jefferson County (Kentucky) Medical Society, Feb. 2, 1925, and the report will eventually appear in the transactions of that organization.

Physical Examination.—The patient was a well developed but rather poorly nourished and somewhat emaciated white man. Polyarthritis was present, several joints being involved; the patient complained of acute pain on manipulation. The skin surface presented a yellowish, parchment-like appearance; the ribs and sternum were prominent at the costosternal margin. The tongue showed a grayish mem-

Fig. 1.—Keratoderma of soles.

brane on the edges; it was raw in the center and beefy red; the tonsils were hypertrophied and protruding; the teeth were in good condition, but several were absent, the result of injury.

The nails of the middle and little fingers of the left hand were absent, and in their place there was a dry, scaly, cornified substance. Paronychia was noted

on the ring finger of the right hand. The soles were dry and tough and contained several discrete papules, pustules and areas of hyperkeratosis.

Considerable inflammation was noted about the external urethral orifice; the left groin was excoriated as were also the perineum and area surrounding the anus. A slight urethral discharge was present. The prostate was somewhat enlarged and tender. On massage apparently purulent material was expressed into the urethra and appeared at the meatus externus. Diagnosis, based on clinical findings, was: "urethritis, polyarthritis and hyperkeratosis," or the "keratodermie blennorrhagique" of Vidal.

Laboratory Findings.—Urinalysis revealed: color, straw; appearance, cloudy; reaction, acid; albumin, + +; sugar, negative. Microscopic examination revealed

Fig. 2.—Lesions of hand and foot.

many pus cells, a few epithelial cells and amorphous urates. Blood examination revealed: erythrocytes, 4,680,000; hemoglobin, 90 per cent.; leukocytes, 16,500. The differential count was: polymorphonuclear neutrophils, 65 per cent.; basophils, 2 per cent.; lymphocytes, 31 per cent.; transitionals, 2 per cent. The feces were negative for parasites. The blood Wassermann reaction was negative.

The accompanying illustrations show the conditions present when the patient was exhibited, Feb. 2, 1925. All the nails of both fingers and toes show the characteristic hyperkeratotic changes. Disintegration always begins in the nail plates at the distal extremities; the nails became much thickened and elevated

because of pressure beneath, brittle and yellowish. Many layers of crust form, which elevate the nails from their beds and cause partial or complete destruction.

The cutaneous lesions began as vesicles, then became pustules, and finally crusting or cornification appeared. The hyperkeratotic areas on the soles and palms were symmetrical. There were a few isolated lesions on the scalp and other portions of the body surface, but the hands and feet are principally affected. During the progress of the disease the palms and soles often present, as in this case, lesions in various stages of evolutionary development, vesicular, pustular and hyperkeratotic areas being found closely associated.

The fact is noteworthy that repeated microscopic examinations of the urethral discharge and material obtained from the cutaneous lesions in this case failed to disclose either Neisserian diplococci or fungi of any type. I believe this has also been the experience of others who have observed similar cases, with the exception noted.

Treatment and Course.—The therapeusis in the foregoing case consisted of routine local and general medication applicable to chronic Neisserian infection. The polyarthritis and also the hyperkeratotic lesions have gradually improved since the institution of vigorous "antigonorrheal" treatment. The patient has less articular pain, many of the cutaneous lesions have completely disappeared, no new ones have developed, and the urethral discharge has subsided. The greatest benefit seems to have been derived from the intravenous introduction of mercurochrome-220 soluble in 1 per cent. solution. Medication applied locally to the hyperkeratotic areas is of uncertain value, but in this case so-called "electric baking," hot saline compresses and the local use of mercurochrome appeared to hasten resolution and healing of the lesions.

The patient continued to improve and was dismissed from the hospital apparently well after eight weeks of treatment.

COMMENT

The splendid article by Keim, to which reference has already been made, contains a complete bibliography of the subject, from 1893 to the beginning of 1923, and shows a total of fifty-nine recorded cases. Adding the cases since reported brings the total to sixty-five.

HISTOPATHOLOGY OF SYPHILITIC HYPOPLASIA OF THE TEETH *

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To obtain a clear concept of the dental stigmas of hereditary syphilis has been the desire of every student of the subject. Some authors claim that dystrophies of certain teeth are among the most important, most persistent and most characteristic lesions produced in the body by hereditary syphilis. Since the time of Hutchinson, many investigations and clinical studies have thrown considerable light on the subject. On the other hand, opposing theories, chiefly proposed by clinicians and not substantiated by much laboratory evidence, have tended to obscure certain pathologic entities in the teeth, and as a result have created a miasma of confusion.

Among such theories was the rachitic theory of Parrot, the eclamptic theory of Magitot, the hereditary theory of Galippe and the toxic theory of Capdepont. As early as 1743, Bunon described the erosion of teeth attributed to rickets, measles and scurvy. The first mention of syphilis in connection with dental alterations was made by Sanchez in 1785. He referred to the changes in the teeth as "black decay," and associated them with mental deficiencies, diseases of the eye and certain other anomalies.

Dental malformation as a diagnostic element in congenital syphilis was first emphasized by Jonathan Hutchinson in 1857, after he had made numerous observations in patients presenting lesions of the eye, of the ear and of the teeth. The characteristic crescent-shaped deformity of the biting edge of the upper incisor was accurately described by him as being pathognomonic of hereditary syphilis. His views were opposed by Magitot, who presented before the Congress in London (1881) a maxillary bone of a cow showing the same lesion on the incisors, which he attributed to eclampsia. In 1884, Henri Moon described the hypoplastic molar of syphilis in a monograph published in "Bryant's Surgery." Three years later, Sollier observed that in many instances dental malformations in idiots and backward children were not unlike those occurring in syphilis. Interest in this subject was subsequently

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* Presented as an exhibit, sponsored by Dr. H. N. Cole, at the Atlantic City Session of the American Medical Association, May, 1925.

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sustained by the French school under the leadership of such clinicians as Fournier and his son, Hallopeau and Darier. The elder Fournier, fortified by a great amount of clinical experience, declared that cuspal hypoplasia of the six-year-molar was as pathognomonic of hereditary syphilis as the Hutchinson incisor; he further maintained that either one or both of these deformities was found in about 40 per cent. of all patients suffering with congenital syphilis. This group he characterized as having hereditary syphilis of direct transmission comparable to acquired syphilis, with distinctly typical lesions. In a second group of doubtful or borderline cases, he found dental manifestations not quite as typical, which he regarded as parasyphilitic or residual characteristics, along with mental retardation, diminution of vital resistance, infantilism, convulsions and general impairment of constitution.

Prior to 1870, few writers had made any study of the histologic characteristics of syphilitic hypoplasia. The first important investigation of this phase was made by Zsigmondy, who presented a paper at the World's Columbian Dental Congress in 1893. Black,[1] the father of American dentistry, was one of the few to apply the knowledge of the embryology of the teeth to the subject of hypoplasia and to make chronologic analyses of such deformities. He was largely instrumental in reviving and stimulating the interest of the dental profession in all forms of hypoplasia. His brilliant monograph, "Atrophy of the Teeth," has not been as accessible to the medical profession and particularly to the syphilologist as it might have been. His knowledge of the chronology of deposition of enamel and dentine—knowledge which is necessary for a complete understanding of the nature of hypoplasia—has been ignored by many in our own ranks who have attempted to crystallize our conceptions of syphilitic teeth.

In 1908, Cavallaro[2] of Florence, Italy, published the first extensive work on dentition in hereditary syphilis. Influenced largely by Fournier, he was led to conclude that practically all forms of dental hypoplasia were due to syphilis, particularly when about the same time Pasini claimed discovery of *Spirochaeta pallida* in the dental tissues of a congenital syphilitic fetus of seven months. Cavallaro studied dissections, microscopic sections and roentgenograms of fetal maxillae, both syphilitic and normal. In his chronologic analysis, he used a table of dentinification of the teeth from which he proceeded to fix the chronology of enamel hypoplasia, and was thus led to conclusions which are at great variance with those of the present work.

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1. Black, G. V.: Pathology of the Hard Tissues of the Teeth, Chicago Medico-Dental Pub. Co., 1914.

2. Cavallaro, J.: Syphilis in Relation to Dentition, Dental Cosmos **50**:11, **51**:1, 1908-1909.

In this country, Stein,[3] before the Section on Stomatology in Philadelphia in 1908, read a paper on the dental stigmas of syphilis. He presented a table of the inception of dentinification of the teeth, and, in the same manner as Cavallaro, drew conclusions as to the chronology of hypoplasia of enamel from such a table. In spite of the researches of Broomell,[4] and of Pierce and Black on the chronology of tooth development, he concluded that cuspal hypoplasia of the six-year molar was entirely prenatal and that cross striations or pits half way up the incisors were due to malnutrition episodes of the first few months of extra-uterine life. Stoll[5] and others who applied Stein's table of the chronology of dentine deposition fell into the same errors in attempting to determine the time element of various hypoplastic striations as seen on the enamel. Only in comparatively recent years has the influence of short intercurrent

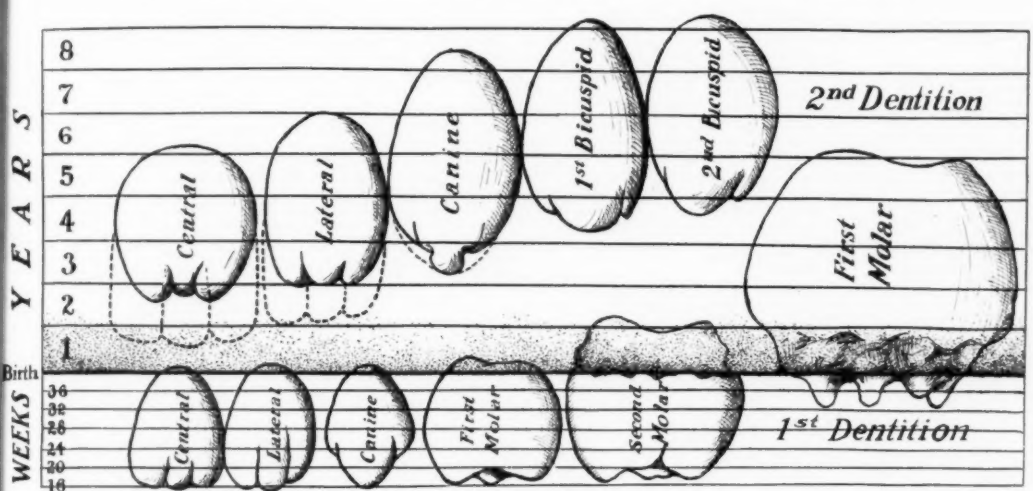


Fig. 1.—Table of inception and duration of enamelization of the first and second dentition, showing the time factor of congenital syphilis and the manner yin which it affects the second deciduous molar, the first permanent molar and the enamel organs of the incisors and the cuspid. The intensity of the stippling in this syphilitic zone represents the degree of severity of effect.

infections been recognized in leaving their impress on the growing teeth. Since exanthems are common between the ages of 3 and 6, such acute episodes can well offer an explanation of the sharply defined hypoplastic zones and honeycomb notches seen on the labial surfaces of anterior

3. Stein, J.: Dental Stigmata of Hereditary Syphilis, Dental Cosmos **55**:695, 1913.

4. Broomell, I. N., and Fischelis, P.: Anatomy and Histology of the Mouth and Teeth, Philadelphia, P. Blakiston's Sons & Company, 1923.

5. Stoll, H. F.: Clinical Diagnosis of Heredo-syphilis, J. A. M. A. **77**:919 (Sept. 17) 1921.

teeth. Such deformities, varied as they are, readily promote the greatest occasion for confusion in recognizing the stigmas which are characteristic of syphilis.

METHODS OF STUDY

As an embryologic study of the manner and the time in which the various zones of the tooth substance are formed is a necessary basis for the correct chronologic analysis of various types of hypoplasia, a preliminary survey of the works of various writers was made. It is evident that if an estimate of the time in which a fault occurred is to be made, a table of calcification must be available. As previously stated, a number of workers have attempted to apply a table of dentinification instead of one of enamelization in the interpretation of hypoplastic deformities. The results of Broomell, Preiswerk,[6] Fischer,[7] Scherbel,[8] and Black on the inception and duration of enamel deposition were used as a basis. These writers have made dissections of both fetal and extra-uterine jaws, sections and roentgen-ray studies, and from their conclusions, a composite table was formulated (Fig. 1). A fairly wide margin of disparity was found in the chronology of the deciduous teeth whose growth is rapid, but the results in the case of the permanent dentition were fairly constant. Particular attention was paid to the development of the second deciduous molars, the permanent central incisors and the first permanent molars, these three, most authorities admit, being the most commonly affected in congenital syphilis. Of striking interest and significance is the fact that only a minute portion of the first permanent molar is actually formed in utero.

The object of the present work was to scrutinize the typical stigmas of syphilis and other types of hypoplasia with which they may be confounded, in order to determine a time factor which might account for the deformities peculiar to congenital syphilis. In the human teeth there is no regenerative process, and the deformity of hypoplasia is a permanent and fairly accurate record of nutritional or pathologic disturbance occurring during the formation of the successive layers of enamel and dentine. The zones of injury in the enamel rigidly follow the growth or incremental lines of Retzius, which show distinctly the order in which the enamel cap is built up, layer on layer until the entire crown is completed. This lamellation begins at the dento-enamel junction and grows from within outward. In like manner the dentine is formed, beginning at the same junction but progressing from without inward toward the

6. Preiswerk, G.: Zahn Heilkunde, München, Lehmanns, 1919.

7. Fischer, G.: Bau und Entwickelung der Mundhöhle des Menschen, Leipzig, 1909.

8. Scherbel, G., and Schoenlank, W.: Leitfaden der Normalen und Pathologischen Histologie der Zahne, Berlin, 1922.

pulp. The growth of dentine is considerably in advance of that of enamel, and, therefore, a table of dentinification is not applicable to the study of layers of enamel.

The material consisted of extracted teeth from both syphilitic and nonsyphilitic patients presenting all types of hypoplasia. Casts of the jaws were first made to show general topography, and sections were ground, according to the method of Chase and Patton,[9] of freshly extracted teeth and of specimens immersed for two weeks in alcoholic fuchsin. Several reconstructions were made of wax from longitudinal sections

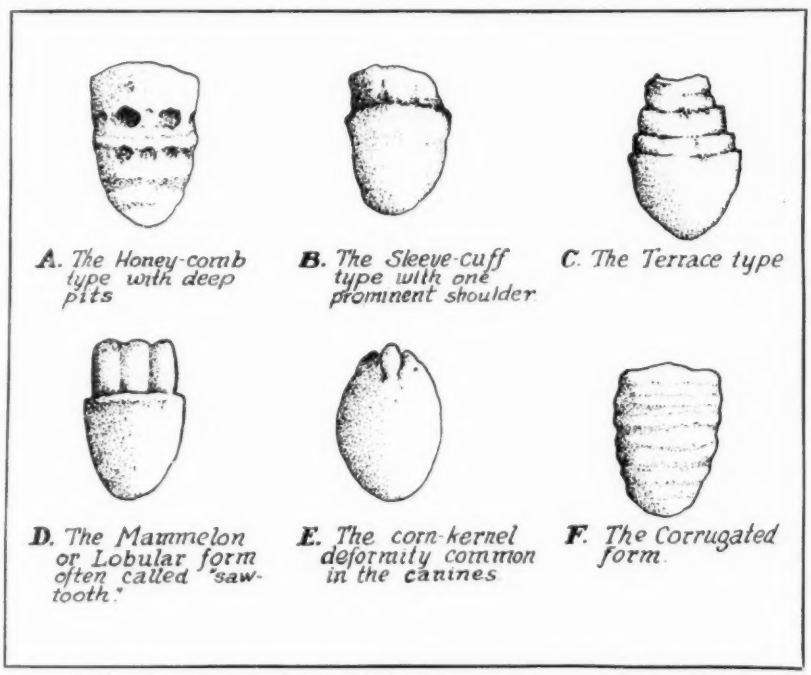

A. The Honey-comb type with deep pits
B. The Sleeve-cuff type with one prominent shoulder
C. The Terrace type
D. The Mammelon or Lobular form often called "saw-tooth."
E. The corn-kernel deformity common in the canines
F. The Corrugated form.

Fig. 2.—Common forms of hypoplasia of the anterior teeth.

HYPOPLASIA OF INCISORS

In a large group of nonsyphilitic patients, a number of fairly representative types of incisor hypoplasia were recognized. In most instances the subjects were children, in whom the pathologic picture was not modified by such factors as abrasion, attrition or caries. Figure 2 presents some of these more common and distinct forms, all of which, with one exception, show some cross striation features. In the majority of persons in whom these deformities were found, a careful history disclosed a

9. Patten, B. M., and Chase, S. W.: Simple Apparatus for Sawing and Grinding Sections of Bone and Teeth, Anat. Rec. **30**:2 (May) 1925.

prolonged or short period of nutritional upset in early childhood. Indigestion, intoxication and disturbed nutritional balance associated with acute infections were elements common to most of these histories. Rickets as a distinct entity was not as frequently demonstrated as had been anticipated.

One such form of hypoplasia with the common "honeycomb" notching, in a tooth extracted from a student, aged 20, is submitted with a chronologic analysis (Figure 3). A low power magnification section presents one zone of injury, about one third of the distance from the incisal edge to the gums. It is made evident that the growth of enamel was arrested along the zones of Retzius, corresponding to about the second year. By studying the photomicrograph carefully, the relations of the enamel zones formed before and after the injury may be made out. It is important to note that the incisal layers are intact. The zones immediately succeeding the deficient layers are subject to a peculiar deformity. They appear curled and apparently when still soft had telescoped into the deficient zones, thus accounting for the pit or sulcus. Such a deformity indicates that the etiologic factor was a fairly acute one of sudden onset and of just as sudden cessation. It can be further noted that a concurrent defect occurred in the dentine, as is indicated by the interglobular spaces along the growth lines in that structure. That such defects are systematic is clinically recognized (Fig. 4). They are always bilateral and occupy the same level on homologous teeth and a different one on teeth of a different order, showing the relative periods of calcification. Any number of recurring upsets may leave their imprints on the coronal surfaces, as demonstrated by numerous cross striation defects of varying degrees of intensity. In all such injuries, the outstanding feature is one of transitory calcium deficiency. In no instance is there a gross deformity of the general shape and size of the tooth. The biting edges of all such teeth are fairly intact and normal in contour. Such aberrations as microdontia (dwarfism), lobular deformities and gross malformations are rather rare, since the injury has not been so extensive as to cause serious shortening or fusion of the various layers, particularly near or at the biting edge.

THE HUTCHINSON INCISOR

A study of this classic deformity presents some entirely different features. The teeth examined were those of children with unmistakable congenital syphilis, but with no apparent modifying nutritional after effects. With such a selection it has been possible to crystallize a distinct type of tooth deformity which presents an interesting embryologic feature. A reconstruction of the pure type of hutchinsonian deformity,

as such, compared with a normal incisor shows that the chief aberration is one of gross deformity of the developmental lobes of the tooth as well as of size. Closer examination shows that the outstanding defect involves the incisal edge of the middle lobe (Fig. 5). All the lobes, in fact, are distinctly undersized at the biting edge, and they seem gradually to arrive at a more normal caliber toward the gum line. As a result the tooth assumes a peg shape and appears as if a hemostat had pinched the middle of the incisor edge while it was still soft, constricting it and allowing the lateral lobes to fall in toward the midline. In no specimen of the series was there any evidence of well-defined cross striations or shoulders. The so-called crescentic notch has repeatedly been emphasized, but in these studies it is not an outstanding feature unless we regard the retraction of the middle lobe as a crescentic deformity. This

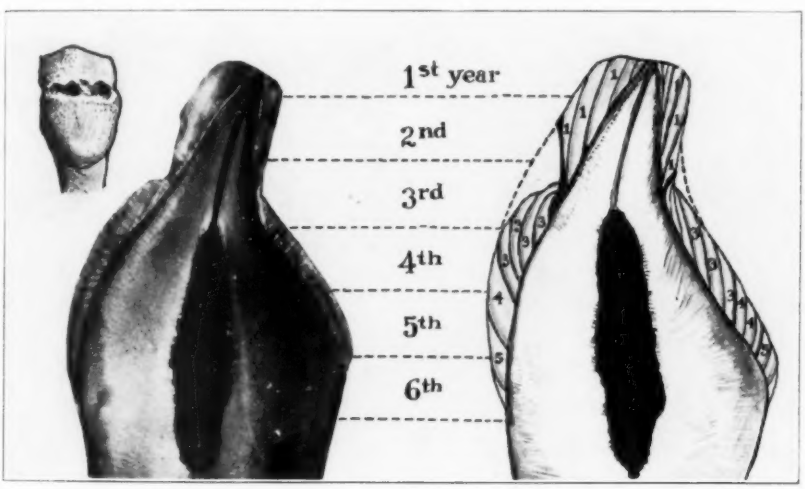


Fig. 3.—Chronologic analysis of an incisor showing a hypoplastic defect in the second year zones.

dimpling may be indistinct, but nevertheless is an important element in differentiating the Hutchinson tooth.

A labiolingual section of such a tooth somewhere through its middle lobe gives one some idea as to the embryologic mechanism of the severe malformation (Fig. 6). The section appears not unlike that of a normal canine with a short crown and a comparatively wide neck. An analysis of the enamel layers discloses that the hypoplastic defects are most severe in the earliest deposits of the enamel and dentine, corresponding to the first year. The condition seems to be less severe in the second year layers and shows a gradual improvement in those succeeding. Here we see the earliest and most essential zones—those of the biting edge—showing irregularities and distortions which induce a maldeposition of

all the subsequent layers. These are more inclined on each other, particularly on the lingual side. Note that there are no deep sulci or notchings on the [illegible] surface. The dentine, even more than the enamel, shows gross [illegible]cies in the earliest layers or caps. These are heavily dotted with interglobular spaces and seem to have telescoped into each other without adding to the length of the crown, thus accounting for a greater part of the shortening of the crown as a whole. It is evident, therefore, that such a tooth, both from the gross and histologic standpoint, is one which was laid down with enamel and dentine organs not only functioning deficiently, but previousy undersized. Certainly the small incisal lobes were the product of a diminutive enamel organ. The greatest aberration of deposit apparently occurred during the first year when about one fifth of the crown was in the process of development.

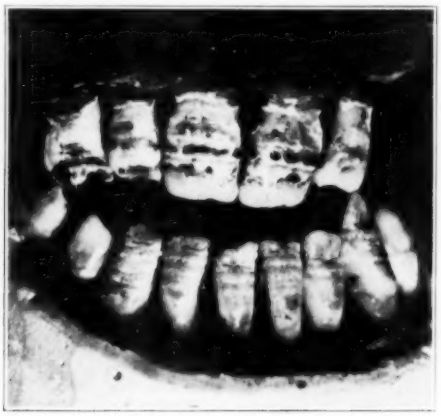


Fig. 4.—A systematic hypoplasia of all the anterior teeth from a patient, aged 28, with a history of "summer colics" and nutritional upsets, recurring annually from the second to the eighth year, with intercurrent infections (measles and whooping cough).

The general picture is one of a fairly intense hypoplasia of the first year gradually improving with each successive layer added for the completion of the crown.

Cavallero attributed many of the common hypoplastic deformities of anterior teeth to syphilis. It is entirely possible that he overlooked the fact that many heredosyphilitic children also suffer with concomitant nutritional disturbances which leave their impress on the teeth and thus mask the original deformity or produce a composite picture. To the lobular defect of Hutchinson is added the cross striation and notching of a fluctuating metabolic disturbance. If this is a common occurrence, one can readily see why such a complex deformity will fail to assist as a reliable element in the diagnosis of congenital syphilis.

THE MULBERRY MOLAR

Hypoplasia of the first permanent molars should receive some special consideration because of its great frequency and because it has been regarded of great significance by many clinicians in the diagnosis of congenital syphilis. The most common deformity produced is one referred to as the mulberry molar of Fournier or Moon. It has been a general opinion that this crumbled and pitted deformity of the occlusal surfaces of the molars occurs because this portion of the tooth is laid down before birth. Both Cavallero and Stein have claimed that such molars, together with the Hutchinson tooth, prove that syphilis is most active just before birth and during the first year. Stein, however, could not explain just why the disease affects only the prenatal portion of the molars, and later, after birth, deforms the central incisors.

An embryologic study of the first molar shows that while calcification of the enamel begins about a month before birth, such a deposit is limited to three or four isolated caps or spiculae, and it is never so

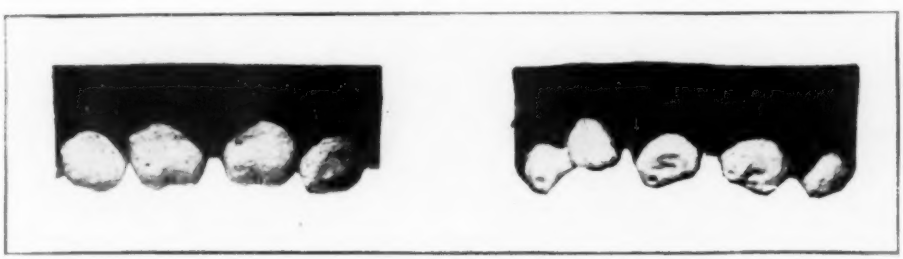


Fig. 5.—Casts of the upper anterior teeth, recently erupted, of two children, aged 7 and 8, respectively, showing the characteristic lobular deformity of the Hutchinson incisor.

advanced at birth that these cusps are well united by calcified layers. It is apparent that these caps, which are the beginning of the true cusps of the molars, while still separated at birth, are nevertheless close together. They are gradually separated and thus determine the caliber of the crown as the infant jaw grows in size. Figure 7 is a section showing the manner in which these "intra-uterine" caps of both dentine and enamel lie in the dental follicle. In gross studies by Broomell and Black, the same observations were made of the status of calcification of the first molar at birth. It is apparent, then, that a hypoplasia of the cusps cannot be attributed to intra-uterine disturbance. A further study of the development of the molar crown shows that the bulk of the occlusal surface is laid down during the first year and that the cusps do not attain their full size until well into the second year. At this time, therefore, calcium deficiencies are likely to produce the greatest degree of distortion. During the very early period of life the successive

layers not only determine the shape of the occlusal lobes and cusps, but "set" these cusps at the proper distances from each other. Hence if hypoplasia occurs very early, or before these cusps are joined together, the cusps themselves will remain closer together than in normal teeth, and the whole of the occlusal layer of the enamel is dwarfed, discolored and sunken into the dentinal crown.

Syphilitic hypoplasia of the first permanent molar, if seen immediately after eruption, is characteristic (Fig. 8). One readily sees why the term "mulberry" furnishes an apt description. The whole occlusal surface is rough, pigmented and pinched, with the exception of two or three nodules of fairly good enamel huddled together which represent the poorly developed cusps. All around this occlusal deformity, the enamel

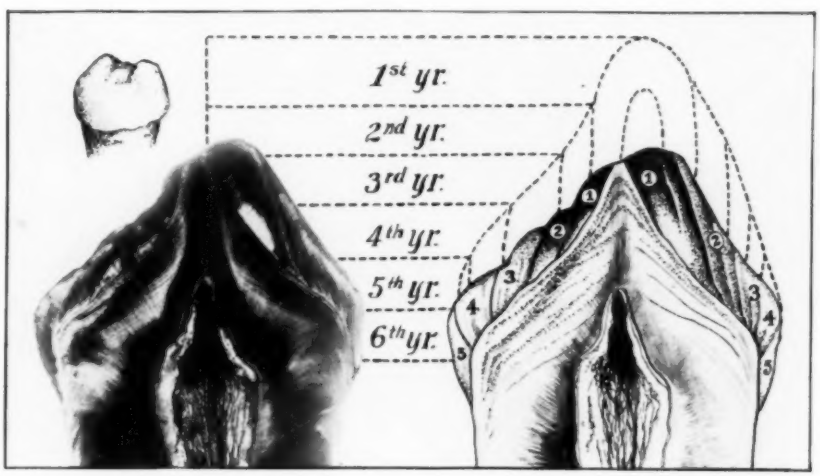


Fig. 6.—Labiolingual section of a syphilitic incisor, with a chronologic analysis, showing the hypoplastic defects in the enamel and dentine deposits of the first and second years. Note the stunted and canine-like appearance of the crown.

of sound formation wells out in the form of a broad collar to the normal size of the crown at the neck of the tooth.

A chronologic analysis of this tooth presents some interesting features (Fig. 9). At first glance, one notes that the greater portion of the grinding surface is irregular, darkly stained and subject to early caries between the cusps. A closer scrutiny shows that "prenatal caps" of enamel are sharply defined and show little or no structural deficiencies. Immediately above these layers, however, the broad lamellae representing the first year's calcification show deeply stained enamel hypoplasia with the greatest intensity at the "birth line" and gradually lessening toward the succeeding second year layers. This sharp contrast between the

deposits made before birth and those immediately after, is demonstrable in all the preparations of syphilitic molars studied, and seems to indicate that the disease has a rather precipitous effect on the active enamel organ as soon as the individual is separated from the placental circulation. There is reason to believe, therefore, that the lesion producing such an abrupt change in the character of the enamel can well be a part of the severe stomatitis and respiratory tract inflammation one sees in the newborn who have active syphilis.

The hypoplasia gradually improves so that the later formations of enamel, particularly those which make up the most distal portions of the cusps and lobes, are of good quality. They are the deposits of the

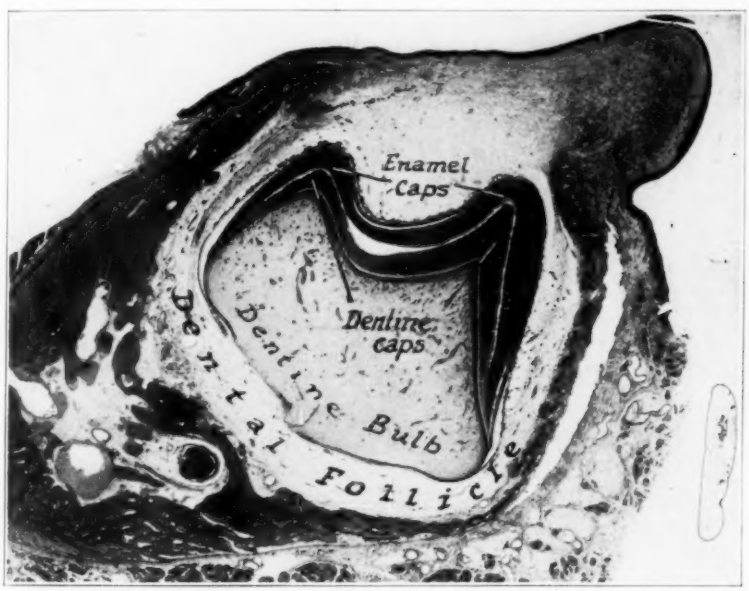


Fig. 7.—Section through the follicle of a developing molar shortly after birth showing the manner in which the primary enamel and dentine layers are related. In this section, two adjacent "caps" are united by both enamel and dentine. Compare these "caps" with those in the fully developed molar.

second year. Grossly such layers appear as white domes on the tips of each cuspal nodule.

Those layers of enamel which succeed the hypoplastic deposits and which are formed from the second to the fifth year are likely to be subject to various maldepositions as they attempt to apply themselves to the deficient zones. A reconstruction (Fig. 10) in wax from three longitudinal sections shows the characteristic deformity of the two cusps lying close together. The succeeding healthy layers are distorted and seem to telescope into one another and bulge outward. The result is the

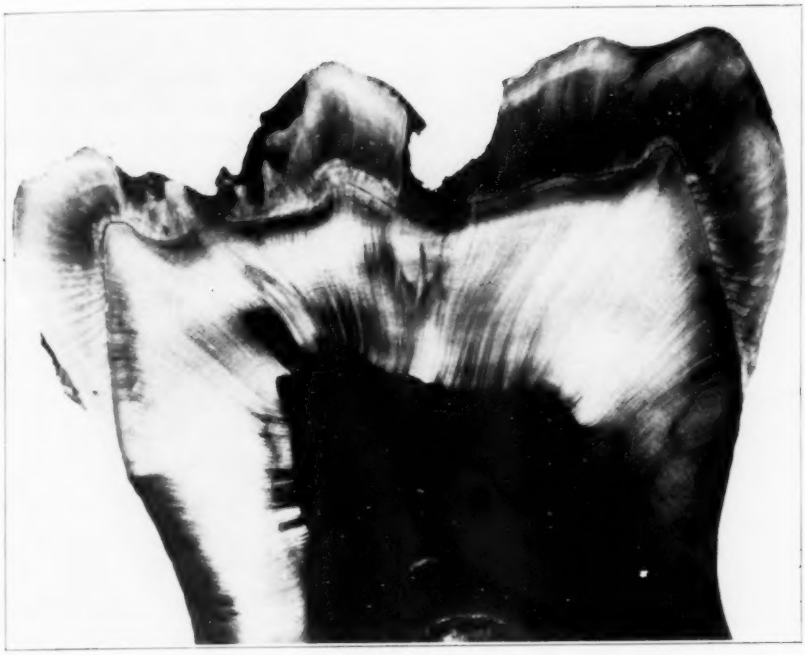
formation of the familiar tubercle or pseudocusp of Carabelli, which some clinicians emphasize as an important feature of the syphilitic molar. In a true sense this structure is not a supernumerary cusp, because it possesses no underlying dental cap. The true supernumerary cusp which generally lies on the lingual aspect of this molar, and which is regarded by the average dentist as an anomaly of no special significance, should not be confounded with the tubercle of hypoplasia. The latter lies, generally, below the level of the normal cusps at the junction of the hypoplastic and normal portions of the crown. It is a feature, consequently, of any severe hypoplasia of the molar in which the first year layers are dwarfed and grossly deficient.

Is this deformed molar a reliable evidence of congenital syphilis? What explanation can we offer for the presence of hypoplastic molars of an apparently similar type in many children who show no other stigmas? Can any other severe nutritional upset of the new-born produce a

Fig. 8.—Three mulberry molars extracted from patients with congenital syphilis. Note the nodules crowded together on a dwarfed and hypoplastic occlusal surface.

lesion of the same kind? These questions have been occasions for frequent and heated controversies. Black takes vigorous opposition to the view of Fournier and Cavallero and claims that other factors which have the same chronology can account for the mulberry molar. He states that case after case was presented to him, at dental clinics, in which syphilis was not even a remote possibility. In a foregoing statement it was emphasized that Fournier regarded such instances as parasyphilitic or residual characteristics.

A number of such persons were presented for study with the idea of determining some finer differentiating elements. While the molars of a few strongly resembled the features of the molar heretofore described, the majority of specimens extracted showed notable deviations.



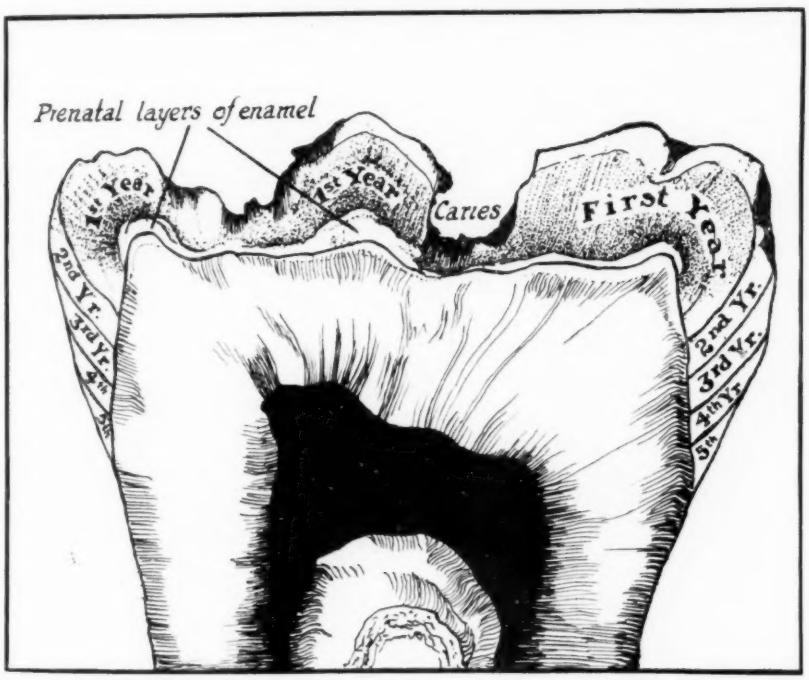


Fig. 9.—Section through a mulberry molar of syphilis with a chronologic analysis, showing the disposition of the hypoplastic enamel in the first year layers. Note the well defined "prenatal caps" which stand out in striking contrast to the deficient layers just above them. The hypoplastic layers are indicated by the deep stippling.

Instead of the pronounced cusps, topped with sound enamel, and bunched closely together, these molars showed smaller elevations, wider apart and with no cuspal dome of white healthy enamel. The biting surface was flatter, with finer irregularities and with a more diffuse discoloration.

A chronologic analysis of such specimens accounts in part for this difference in deformity. The hypoplasia is found in the later zones, that is, at the end of the first year or well in the second year layers, and, therefore, conforms to the cross striation lesions of the anterior teeth as described under the heading of nonsyphilitic hypoplasias. Figure 11 offers a section study of a syphilitic molar as such, along with one in which the deformity presents the features just mentioned. The latter tooth, while hypoplastic, does not quite fulfil the description of a mulberry tooth, and therefore does not fall in that category.

This comparison does not pretend to establish a reliable working differentiation. Mention has already been made of the possibility of a nonsyphilitic disease having the same time factor as syphilis and producing an identical molar lesion. However, it can be assumed that the molar presenting a discrete hypoplasia in the second year zones is not likely to be one due to congenital syphilis, provided the premise is acceptable that the time of the latter condition is at the beginning of the first year.

In the first section of this figure one recognizes the mulberry nodules projecting prominently from the occlusal surface of a molar in a case of congenital syphilis. The analysis demonstrates a hypoplasia of the first year deposits of such nature that the proper separation of the cusps has not been affected. The overlying second year layers are sound in quality but are distorted in an attempt to apply themselves to those preceding them. Here one can again appreciate the nature of the pseudocusp formed by the buckling of aftercoming layers.

In the second part of the same figure is presented a molar with distinctly different clinical and embryologic features. The molar was removed from a boy, aged 13, who showed a well defined transverse pitting on the front teeth in approximately the second year zones. There was no clinical or serologic evidence of syphilis. All four first molars present occlusal surfaces of an irregular, serrated nature with poorly defined shallow cusps. There are no mulberry nodules or supernumerary tubercles. The crown is broad at the grinding surface as well as at the neck. A section reveals a hypoplasia of those layers which form the outermost portions of the grinding surface. The first year deposits which are assailed in congenital syphilis are intact, and the cusps themselves are set at the proper distances from each other over dentinal caps which are well proportioned. Here, therefore, one notes a severe zonal

hypoplasia but no pronounced dwarfing of the crown as a whole, nor any remarkable aberration of the healthy aftercoming layers of the third, fourth and fifth years.

This differentiation as offered between two distinct types of occlusal hypoplasia of the molars is, therefore, one based on a difference in the time factor of the disease. A small number of molars submitted for analysis have disclosed that this time factor may be one of both the first and second years, and consequently the tooth shows a composite deformity. It is easy to conceive of a nutritional disturbance which will reflect

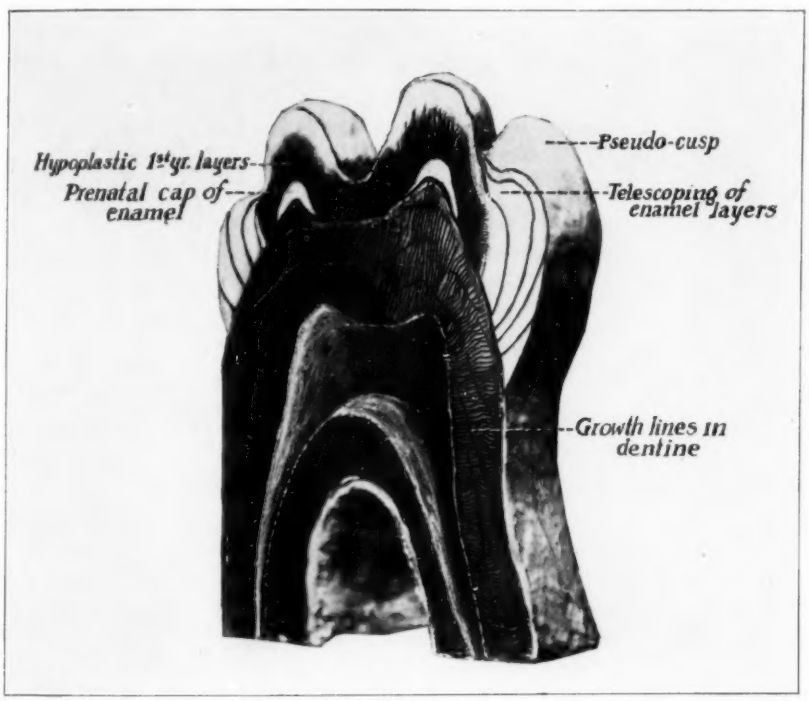


Fig. 10.—A reconstruction of a syphilitic molar, in perspective, showing the two cusps crowded together on a small crown surface and the after-coming layers telescoped to form an "extra-tubercle."

itself on all teeth forming during the first and second years of life without remission. Hence one meets with a severe systematized hypoplasia involving a large portion of the incisal surface of the anteriors including the cuspid as well as the first molars. That congenital syphilis or its immediate after-effects can be included among such disturbances is without question, and, consequently, the analysis of such a composite hypoplasia will not afford a means for a differential diagnosis.

THE DECIDUOUS TEETH

While many writers have described various anomalies and deficiencies of the deciduous teeth as being a part of the picture of congenital syphilis, no one has as yet been able to present a clearly defined aberration which can be relied on as a fairly constant feature. Fournier has described a central incisor in a few isolated cases in the first dentition which resembled the hutchinsonian tooth. It is obvious that if a well crystallized conception of the lesions of congenital syphilis in the first dentition were possible from the diagnostic standpoint, it would be of a decidedly greater value than one of the second dentition. A review of the expressions of many writers fails to disclose as reliable a diagnostic key in the milk teeth as one meets in the permanent dentition. Black, Cavallero, and

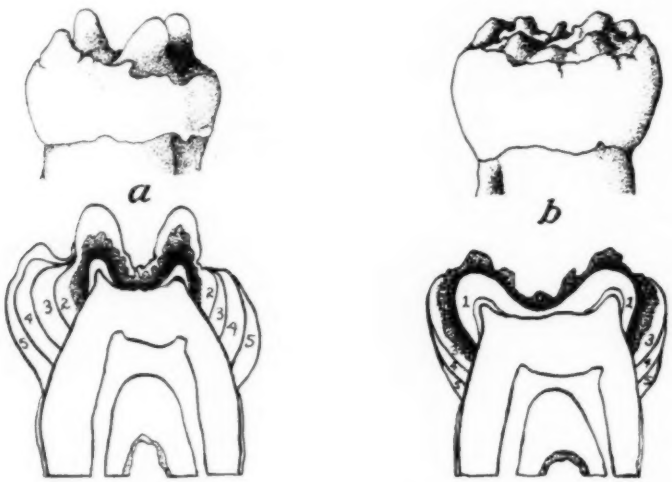


Fig. 11.—A comparison and a chronologic analysis of two kinds of hypoplastic molars. Fig. *a* is that of a syphilitic molar showing the peculiar crowding of the cuspal nodules and the dwarfed crown. The section reveals a deficiency of the first year layers. Fig. *b* is a sketch of a nonsyphilitic molar, an analysis of which discloses a hypoplasia in the second year zones only. Note the normal circumference of the crown in spite of its granular and irregular occlusal surface.

Burchard [10] agree that such general disturbances as delayed eruption, malpositions, early caries and tardy absorption of the temporary roots are common features in congenital syphilis; but all admit that these abnormalities are not sufficiently typical to afford a differential diagnosis. Perhaps the only consistent sign is a hypoplasia of the second deciduous molar. Here the feature most commonly noted is a pitting across the

10. Burchard, H. H., and Inglis, O. E.: Dental Pathology, Philadelphia, Lea & Febiger, 1915.

crown and generally proximal to the occlusal surface. A glance at the table of calcification will show why this tooth, the last to form in the deciduous formula, is thus affected. It is actively in the process of calcification just before and after birth and, therefore, is subject to the same process coming on immediately after birth when the enamel organ of the central incisor and of the first permanent molar is being affected. Only six cases which demonstrated this abnormality were available for study. Two of these children were under observation for a long enough time to verify the suspicion of congenital syphilis on the eruption of typical Hutchinson incisors. The hypoplastic zones in the second deciduous molar of syphilis may be disposed anywhere along the crown, although in three cases out of six, the lesion was found to be just distal to the neck of the tooth.

A chronologic analysis of this molar and of the first permanent molar, part of both being laid down before and part after birth, does not sustain the claim that syphilis of the teeth is active in intra-uterine life. On the contrary, it seems to demonstrate that the tooth-forming organs are not seriously impaired in utero and that most of the deciduous teeth as well as the prenatal caps of the first permanent molar are fairly well formed, in spite of the presence of active syphilis in the fetus. An attempt to explain the mechanism of this seeming paradox would only lead one into the mazes of an immunologic problem.

CONCLUSIONS

1. The true hutchinsonian incisor is a deformity of the developmental lobes of the permanent central incisor and not one of a transitory calcium deficiency. It shows no distinct cross striations.

2. The hutchinsonian incisor can be modified by concurrent nutritional disturbances, causing the formation of a composite lesion.

3. The true mulberry molar of syphilis is a first permanent molar characterized by enamel cusps showing crests of sound enamel on a base of hypoplastic deposits. These cusps are generally crowded together on a crown surface of dwarfed dimensions.

4. The so-called "extra tubercle" on the mulberry molar of syphilis is formed by the buckling or telescoping of normal enamel layers over dwarfed and hypoplastic formations. While it may be a common feature of the syphilitic molar, this structure can be found in any severe hypoplasia of the first year layers. This tubercle of Carabelli should not be confounded with the true supernumerary cusp of the first molar which is an anomaly of no special significance.

5. A hypoplastic molar most easily confounded with the mulberry molar of syphilis is one which shows a rough, granular or serrated

occlusal surface but which otherwise presents a crown of normal caliber. It is a molar of transitory calcium deficiency.

6. With the exception of the second deciduous molar, no well defined lesions of the first dentition are found which can be regarded as deformities characteristic only of congenital syphilis.

7. A time factor of congenital syphilis as a cause of dental hypoplasia seems to be established in the first year of life, the effects coming on suddenly at birth and being concurrent with the stomatitis, rhinitis and the other familiar symptoms of syphilis in the new-born.

GOLD COMPOUNDS IN TREATMENT OF EXPERIMENTAL TUBERCULOSIS OF SKIN IN ANIMALS

A PRELIMINARY REPORT *

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Paracélsus, in 1500, compounded an "elixir of life" made up of gold and mercury, and the use of gold in tuberculosis probably dates back to this time. In 1810, Chrestien, according to White,[1] reintroduced the use of gold in tuberculosis. The use of compounds of gold in the treatment of tuberculosis had, however, its scientific inception in the statement of Robert Koch before the Tenth International Congress of Berlin in 1890. He stated that various salts of gold, especially potassium-gold cyanid, exercised an extremely injurious effect on the tubercle bacillus, preventing its growth in dilutions of 1:1,000,000. Von Behring [2] confirmed Koch's findings, but found that in blood serum the bacteriostatic effect of gold salts was considerably reduced, owing to the presence of the globulins.

Bruck and Glück [3] were the first in recent years to institute gold treatment in tuberculosis. These investigators tried the effect of intravenous injections of potassium-gold cyanid in lupus vulgaris. They state that "there is no doubt that by intravenous injection of potassium-gold cyanid, without local treatment, a striking improvement in lupus lesions is produced." This is justified by the photographs published by them, which leave no doubt as to the demonstrably favorable effects on the cutaneous lesions. No complete cures were effected, however. The influence was increased when tuberculin injections were associated with the gold treatment.

Oberstadt [4] likewise obtained good results in cutaneous lesions, but no complete or perfect cures.

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* From the Research Institute of Cutaneous Medicine.

1. White: Am. Med. Surg. Bull. **7**:388, 1894.

2. Von Behring, quoted by Bruck and Glück: München. med. Wchnschr. 1913, No. 2.

3. Bruck and Glück: München. med. Wchnschr. **60**:57, 1913.

4. Oberstadt: Internat. Zentralbl. f. d. ges. Tuber. Forsch. **40**, No. 1, 1917; abst., Am. Rev. Tuberc. **1**:737, 1918.

Junker [5] reported good results from the use of salts of gold in pulmonary and skin tuberculosis in human subjects. Poor and Geber [6] also obtained good results in the treatment of tuberculosis of the skin with potassium-gold cyanid.

Spiess and Feldt, in a series of papers published separately and in collaboration, between 1912 and 1916, reported on the use of various gold combinations in tuberculosis. Feldt [7] found that tubercle bacilli were restrained in culture by colloidal gold in dilutions of 1 : 1,000,000 and by potassium cyanid in a dilution of 1 : 1,000. He regarded potassium-gold cyanid as too toxic a preparation for human use. As cantharidin earlier had been found to produce focal reactions in tuberculous tissue; this substance was condensed with ethylenediamin and combined with cyanid of gold. The toxicity of this monocantharidyl-ethylenediamin-aurous cyanid, which was called "Aurocantan," was much less than the cyanid of gold and potassium. Spiess and Feldt claimed that the favorable effects were due partly to the influence of the compound on the tubercle bacillus and in part to the stimulation of the defensive mechanism of the body.

In 1917, Feldt [8] elaborated another gold compound, the 4 amino-2 auro-thiophenol-carbonic acid, to which was given the name "Krysolgan." This possesses a tuberculin-like effect on tuberculous tissues, and is said to restrain growths of tubercle bacilli in a concentration of 1 : 1,000,000. During the past seven years it has been extensively employed in Germany in cutaneous and other forms of tuberculosis. No curative effect on tuberculosis in rabbits could be demonstrated by Gorke or Töppich [9] with "Krysolgan," and Kolle and Schlossberger [10] were able to show only some retardation of death. "Krysolgan" has been considerably employed in lupus erythematosus, a disease which many authors consider to be intimately related with, if not due to, tuberculosis.

Martinstein of Breslau appeared to be the first to employ this drug in lupus erythematosus. Ullmann [11] reports successful results with this preparation in eight cases of lupus erythematosus. In a few of the cases tuberculin was also administered, and in some an organic iodin compound. In discussing this paper before the Vienna Medical Society,

5. Junker: München. med. Wchnschr. **60**:1376, 1913; Beitr. klin. Tuberk. **7**: 421, 1914.

6. Poor and Geber: Deutsch. med. Wchnschr. **39**:1303, 1913.

7. Feldt: Deutsch. med. Wchnschr. **40**:579, 1914; Berl. klin. Wchnschr. **82**:365, 1915; München. med. Wchnschr. **61**:842, 1914; **61**:1455, 1914.

8. Feldt: Berl. klin. Wchnschr. **54**:1111, 1917; München. med. Wchnschr. **67**:1500, 1920; Therap-Monatsh. **32**:241, 1918.

9. Gorke and Töppich: Beitr. z. Klin. der Tuberk. **53**:132, 1922.

10. Kolle and Schlossberger: Ref. Zentralbl. f. d. ges. Tbkunde. **19**:341, 1923.

11. Ullmann: Wien. klin. Wchnschr. **37**:579, 1924.

Nobel was unable to substantiate the claims of Ullmann. Volk agreed with Ullmann as to its beneficial effects. He had treated twenty-seven patients and had had rapidly favorable results, particularly in the chronic hyperkeratotic forms. Gruss, of Finger's Clinic, said that he had seen favorable results only in the early stages of lupus erythematosus, but with the combination treated with "Mirion" (an organic iodin compound) he had secured cures in the markedly infiltrated cases. In lupus vulgaris thus far "Krysolgan" seems to have achieved little. Rickman [12] refers to a new gold preparation more recently elaborated in Germany under the name of "Triphal." He says the effect of this drug given intravenously in tuberculosis of the larynx and in combination with other methods can no longer be contested. No definite judgment is as yet at hand with regard to its value in tuberculosis of the skin.

As lupus erythematosus is a most intractable as well as disfiguring disease, the above reported results would appear to be encouraging.

In 1924, Møllgard of Copenhagen published a book on "The Chemotherapy of Tuberculosis," in which he elaborated the virtues of a perfected compound of gold with thiosulphuric acid, which was first produced by Fordos and Gélis [13] in 1845. A pure body with constant chemical and physical properties can be made by way of synthesis indicated by Fordos and Gélis, but it is necessary to recrystallize it four or five times from a mixture of alcohol and water. The yields are poor, and the final product contains only from 10 to 15 per cent. of the gold originally employed. By Møllgard's [14] method the product contains from 90 to 95 per cent. of the gold employed after one crystallization. This product, to which the name "Sanocrysin" has been given, is snow white, occurs in long needles, and is easily soluble in water, with an almost neutral reaction. It is slowly decomposible and does not precipitate serum even at 40 C. in twenty-four hours. It is not supposed to form gold ions in the body. It retards the growth of the tubercle bacillus in a concentration of 1 : 1,000,000, and prevents it in 1 : 100,000.

The minimal lethal dose of Sanocrysin in guinea-pigs is 30 mg. per kilogram given intraperitoneally, and intravenously in rabbits the minimal lethal dose is 40 mg. per kilogram. Calves tolerate intravenously 60 mg. per kilogram. Møllgard makes the following experimental claims for Sanocrysin: (1) It prevents growth of tubercle bacilli in culture. (2) It makes the fatty system which protects the tubercle bacilli penetrable to hydrogen and metal ions. (3) It prevents infected guinea-pigs from getting generalized tuberculosis. (4) Injected into the tuberculous organism, it produces all the reactions which are expected

12. Rickman: Münch. med. Wchnschr. Nov. 14, 1924, p. 1609.

13. Fordos and Gélis: Ann. Chem. Phys. **13**:394, 1845.

14. Møllgard: The Chemotherapy of Tuberculosis, Copenhagen, Busck, 1924.

to follow the injection of a substance having a bactericidal influence on tubercle bacilli. (5) Injected into nontuberculous organisms in therapeutic doses it produces no reactions.

It should be here remarked that the guinea-pigs inoculated subcutaneously immediately received injections with Sanocrysin in 10 to 20 mg. doses *into the infected area.*

Favorable results in various forms of human tuberculosis are claimed.

Our interest in the use of gold compounds in tuberculosis was primarily stimulated by observing that of twenty-two salts of metals and metalloids which we brought into contact in solution in the test tube with given quantities of tubercle bacilli, gold exhibited the highest combining power. (This study is being published elsewhere.) Further investigations, however, indicated that this affinity was general rather than specific for the bodies of bacteria and organic tissues.

EXPERIMENTS WITH VARIOUS GOLD COMPOUNDS

We have elaborated a number of gold compounds which we have tested in animals inoculated with the human and bovine strains of the tubercle bacillus. We first employed a sodium-gold-arsphenamin produced for us by Dr. George W. Raiziss of Philadelphia. This was tolerated intravenously by white rats in the dose of 90 to 100 mg. per kilogram.

We subsequently employed on seventeen guinea-pigs the following gold compounds produced in our institute: gold naphthol blue, gold-hexamethylenamin-thiosinamin, gold hexamethylenamin-thiocarbamid, gold and sodium thiosulphate and a gold protein compound. The gold and sodium thiosulphate we believe to be identical with or closely allied to that elaborated by Møllgard.

Guinea-Pigs Treated with Sodium and Gold-Thiosulphate (G22) and a Gold-Protein Compound (G23).—In the following experiment, six guinea-pigs were infected subcutaneously with 0.01 mg. of bovine tubercle bacillus. Twenty-four hours later, three of them were given an injection intraperitoneally of 5 mg. per kilogram of G22 (gold and sodium thiosulphate) and the other three 10 mg. per kilogram. After receiving twelve doses, owing to loss of weight, treatment was instituted with G23, a gold-protein compound. Of the latter they received from one to eight doses. Two guinea-pigs were infected but not treated, and two were treated but not infected, the latter being kept as drug controls. The infected and treated animals all died of generalized tuberculosis in from forty to sixty-seven days. There was no prolongation of life as the infected controls lived sixty-seven and seventy-seven days, respectively. The two drug controls survived.

These experiments confirm all previous unsuccessful attempts to prevent tuberculosis in guinea-pigs when the infection is by the subcutaneous route and the drug administration by intraperitoneal injection. Møllgard, as has been stated, protected guinea-pigs with "Sanocrysin"

TABLE 1.—*Treatment of Guinea-Pigs with Various Gold Compounds*

Infective Dose Tbc. Bovine	Compound	Interval Between Infection and Treatment, Hours	Dose Gm. per Kilogram	Total Doses	Total Amount, Gm.	Average Interval Between Treatments, Days	Died or Killed	Result of Treatment as Determined at Necropsy
1 mg. subcutaneously	Gold-naphthol-blue	24	0.005	7	0.0117	4	Killed 28 days	Compound had no apparent effect
1 mg. subcutaneously	Gold-naphthol-blue	24	0.005	7	0.0192	4	Killed 28 days	Compound had no apparent effect
1 mg. subcutaneously	Gold-hexamethylenamin-thiosinamin	24	0.005	9	0.6108	Approx. 3	Dead 26 days	Generalized tuberculosis
1 mg. subcutaneously	Gold-hexamethylenamin-thiosinamin	24	0.005	9	0.0131	3.1	Dead 28 days	Generalized tuberculosis
1 mg. subcutaneously	Gold-hexamethylenamin-thiosinamin	24	0.008	9	0.0174	3.3	Dead 30 days	Generalized tuberculosis
1 mg. subcutaneously	Gold-hexamethylenamin-thiosinamin	24	0.008	9	0.0167	3	Dead 27 days	Generalized tuberculosis
1 mg. subcutaneously	Infected control	—	—	—	—	—	Dead 32 days	Generalized tuberculosis

It was observed that 0.008 gm. per kilogram of gold hexamethylenamin-thiosinamin affected the weight materially in the untreated control animal.
At this time the infective dose of the tuberculous bovine strain for guinea-pigs was determined, and it was found that 0.01 mg. of moist tubercle bacilli produced subcutaneous lesions in 15 days.

0.01 mg. subcutaneously	Gold-hexamethylenamin-thiosinamin	24	0.005	4	0.004	2.7	Dead 11 days	No lesions of tuberculosis except at site of inoculation
0.01 mg. subcutaneously	Gold-hexamethylenamin-thiosinamin	24	0.005	6	0.0087	4.1	Dead 25 days	Generalized tuberculosis
0.01 mg. subcutaneously	Gold-naphthol-blue	24	0.005	7	0.0092	3.7	Dead 54 days	Generalized tuberculosis; lived 10 days longer than control pig
0.01 mg. subcutaneously	Gold-naphthol-blue	24	0.005	6	0.0072	5	Dead 30 days	Generalized tuberculosis
0.01 mg. subcutaneously	Gold and sod. chl.	24	0.005	7	0.0097	3.7	Dead 42 days	Generalized tuberculosis; died 4 days before control pig
0.01 mg. subcutaneously	Gold and sod. chl.	24	0.005	6	0.0091	4.1	Dead 25 days	Generalized tuberculosis
0.01 mg. subcutaneously	Gold-hexamethylenamin-thiocarbamide	24	0.003	6	0.0042	5.1	Dead 31 days	Generalized tuberculosis
0.01 mg. subcutaneously	Gold-hexamethylenamin-thiocarbamide	24	0.003	6	0.0048	5.1	Dead 31 days	Generalized tuberculosis
0.01 mg. subcutaneously	Sodium gold arsphenamin	24	0.010	4	0.0061	2.7	Dead 11 days	No lesions of tuberculosis except small abscess at site of inoculation of tubercle bacilli
0.01 mg. subcutaneously	Sodium gold arsphenamin	24	0.010	7	0.0126	3.7	Dead 57 days	Generalized tuberculosis; lived 23 days after average life of infected controls
0.01 mg. subcutaneously	Sodium gold arsphenamin	24	0.005	7	0.0075	3.7	Dead 60 days	Generalized tuberculosis; lived 26 days after average life of infected controls

(sodium and gold thiosulphate) but the injections were given immediately after infection at the site of the introduction of the bacilli. The dose was from 10 to 20 mg. per kilogram, the highest tolerated dose in this animal being 30 mg.

The injection of a virulent strain of bovine tubercle bacilli into so susceptible an animal as the guinea-pig constitutes a most severe test, perhaps too rigorous to determine the value of remedial agents.

Experiments on Rabbits.—We then endeavored to mitigate the infection by employing rabbits instead of guinea-pigs, and furthermore by using *intradermal* instead of *subcutaneous* inoculations.

TABLE 2.—*Effect of G22 and G23 (Combined Treatment) on Experimental Tuberculosis of Guinea-Pigs Infected Subcutaneously*

Guinea-Pig*	Aver. Dose of G22,† Gm. per Kg.	Interval Between Infection and Treatment, Hours	Total No. of Doses of G22	Average Interval Between Injections of G22, Days	Total Amt. of G22 Injected, Gm.	Total No. of Doses of G23‡	Average Dose of G23 Gm.	Total Amount of G 23 Injected, Gm.	Average Interval Between Injections of G23. Days	
47	0.005	24	12	2.9	0.0186	5	0.0275	0.0382	3.3	Dead, 53 days; generalized tuberculosis
48	0.005	24	12	2.9	0.0226	8	0.0275	0.0796	3.3	Dead, 60 days; generalized tuberculosis
49	0.005	24	12	2.9	0.0218	8	0.0275	0.0719	3.3	Dead, 64 days; generalized tuberculosis
50	0.010	24	12	2.9	0.0330	8	0.0275	0.057	3.3	Dead, 67 days; generalized tuberculosis
51	0.010	24	12	2.9	0.0436	7	0.0275	0.0577	3.3	Dead, 62 days; generalized tuberculosis
52	0.010	24	12	2.9	0.0422	1	0.020	0.0054	3.3	Dead, 40 days; generalized tuberculosis
53	Infected control	—	—	—	—	—	—	—	—	Dead, 67 days; generalized tuberculosis
54	Infected control	—	—	—	—	—	—	—	—	Killed, 77 days; generalized tuberculosis
55	0.005	—	12	2.9	0.0206	8	0.0275	0.0936	3.3	Apparently normal, 77 days
56	0.010	—	12	2.9	0.0412	8	0.0275	0.0917	3.3	Apparently normal, 77 days

* Infected, Feb. 5, 1925, with 0.01 mg. moist B. tuberculosis, bovine, injected subcutaneously, except Guinea-pigs 55 and 56 (compound controls).
† G22 was composed of sodium and gold thiosulphate.
‡ G23 was a gold protein compound.

Four rabbits were inoculated intradermally with 0.05 to 0.1 c.c. of a suspension of bovine tubercle bacillus representing 0.01 mg. of the organism. Six punctures were made in each animal. Inflammatory papules developed which grew to nodules 1 cm. in diameter. These ulcerated and became covered with crusts.

Tuberculosis of the Skin of Rabbits Treated with Gold and Sodium Thiosulphate and a Gold Protein Compound.—In this experiment two rabbits, No. 398 and No. 394, were used. On Oct. 13, 1924, they were inoculated by scarification of the skin of the left ear, and a suspension of tubercle bacilli was rubbed in with a sterile swab. The same suspension was injected intradermally into the right ear in six places. Each ear developed well-marked lesions of tuberculosis in both rabbits within six weeks, but the health of the animals appeared to be unimpaired. The lesions produced by intradermal injection persisted, but those produced by scarification gradually disappeared within ninety days. This was true of both rabbits.

On Jan. 15, 1925, Rabbit 398 was placed on treatment by intravenous injections of sodium and gold thiosulphate. This was ninety-four days after infection. A slight improvement was observed eleven days after the first treatment, compared with the control rabbit. A marked improvement in this animal was observed within twenty-eight days, as shown in Figure 1, compared with the photograph of control Rabbit 394 (Fig. 2). This rabbit (398), treated with sodium and gold thiosulphate, and later with a gold protein compound, died on April 22, 1925, 191 days after infection and ninety-seven days after starting treatment. The following is the detailed history of injections of compound, etc.:

Figure 1

Figure 2

Fig. 1.—Rabbit 394 received B. tuberculosis by bovine intradermal infection in ear. Condition is of one hundred and seventy-six days' standing. Animal was untreated control of Rabbit 398.

Fig. 2.—Rabbit 398, after eighty-two days of treatment, B. tuberculosis, bovine infection. It received intradermal inoculations into ear.

Total number of injections of G22, 21.
Total amount of G22 injected, 0.5518 gm.
Average time between injections of G22, 2.9 days.
Total number of injections of gold protein compound, 9.
Total amount of gold protein compound injected, 0.426 gm.
Average time interval between injections of gold compound, 3.7 days.
Total number of injections of both compounds, 30.

Average interval between injections of both compounds, 3.3 days.

Average single dose of G22 amounted to 0.013 gram per kilogram.

Average single dose of gold protein compound amounted to 0.0288 gram per kilogram.

Fig. 3 (Rabbit 66).—Infected intradermally with B. tuberculosis bovine and treated with G. 22 and G. 23. Observe that in some of the lesions the surrounding infiltration has disappeared; one hundred days' duration.

Fig. 4 (Rabbit 67).—Untreated control; infected intradermally with B. tuberculosis, bovine. Distinct annular infiltration about all of the sites of inoculation; subcutaneous tuberculoma present; one hundred days' duration.

Necropsy of Rabbit 398 (Treated Rabbit).—There was some induration at the sites of infection in the skin of the ear, but there was a marked difference between this rabbit and the control (No. 394). There was no observable infection of the lymph glands except in the bronchial and the mediastinal. There were mottled

areas of caseation in the lungs, together with many small foci. One small tubercle was observed in the pleura. No evidence of infection appeared in the liver. The spleen was small and apparently normal. The omentum was apparently normal in appearance. Several sections of the large intestine were studded with many tubercles the size of a pinhead and larger. There were several tubercles the size of a pinhead and larger in each kidney.

Necropsy of Rabbit 394 (Untreated Control).—This rabbit was alive at the time of the death of treated Rabbit 398, but was in poor physical condition, and in order to study and compare the progress of the disease in both animals it was killed.

No apparent macroscopic lymph gland infection was present except in the bronchial and mediastinal lymph glands. Numerous small tubercles covered the whole surface of both lungs; they also appeared throughout the lung tissue. The mediastinum exhibited two small (size of lead shot) tubercles. The pleura showed about twenty fair-sized, flat, caseated areas, approximately 2 to 4 mm.

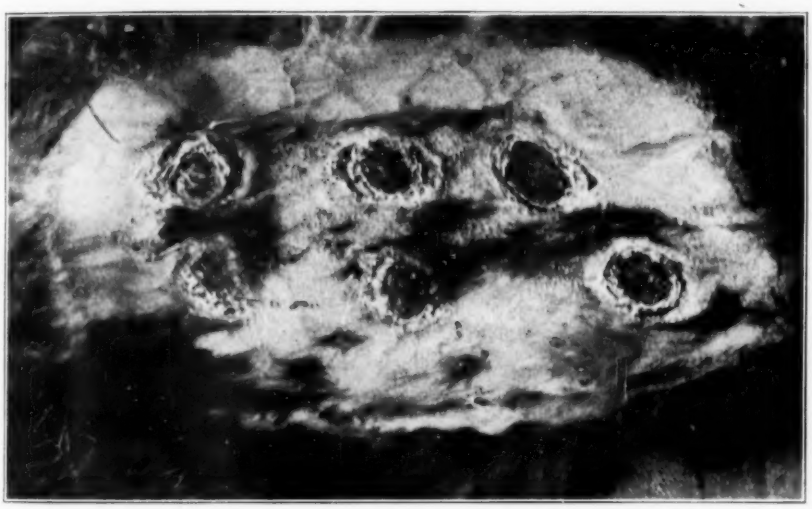

Fig. 5 (Rabbit 67).—Untreated control. Larger photograph showing the type of infiltration around the lesions; one hundred days' duration.

There was no evidence of tuberculosis in the liver. The spleen was small, dark brown and apparently normal. The omentum was apparently normal in appearance. A section of the large intestine 2 cm. long showed many small tubercles. Both kidneys were studded with many tubercles the size of a pinhead and larger.

Comparison of Rabbit 398 (Treated) with Rabbit 394 (Untreated).—The lungs were involved approximately in the same degree. There was more marked involvement of the pleura in the control than in the treated rabbit. The control rabbit showed lesions in the pericardium; the treated rabbit showed none. There were no lesions in the liver, spleen and omentum in either animal. There were considerably more tubercles in the intestines in the treated rabbit than in the untreated rabbit. There

were many times the number of tubercles in the control rabbit than there were in the treated rabbit.

It was noted that the untreated animals *exhibited a great amount of neoplastic infiltration around and beneath the lesions.* One of the latter animals presented a subcutaneous tuberculoma 2 cm. by 1 cm. in diameter.

Scarification Experiments in Rabbits.—Five rabbits were inoculated by scarifying an area 5 by 7 cm. on the back and rubbing in a suspension of bovine tubercle bacillus containing 1 mg. of the organisms to 1 c.c. The lines of scarification

TABLE 3.—*Therapeutic Properties of Gold and a Gold Protein Compound in Tuberculosis of the Skin in Rabbits* *

Rabbit†	Compound Used in Treatment	Interval Between Infection and Treatment, Hours	Total Amt. of G23‡ Injection, Gm.	Total Amt. of G22§ Injection, Gm.	Average Dose of Compound, Gm. per Kg.	Average Interval Between Doses, Days	Total No. of Doses of G23	Total No. of Doses of G22	Weight, Gm.	
63	G23	24	0.076	—	G23 0.010	2.6	3	—	2/26/25 2,615	Died of abortion 12 days after institution of treatment; no tuberculosis
68	G23	24	0.849	—	G23 0.021	3.5	19	—	2/26/25 1,900 5/6/25 2,160	Died, 76 days; died within 15 minutes after 19th injection; tuberculosis of lungs at necropsy
65	G22	24	0.737	0.256	G23 0.0286 G22 0.0125	3.5	10	8	2/20/25 2,800 4/23/25 2/620	Died, 4/23/25, 63d day; tuberculosis of lungs, intestines and kidneys; no tuberculosis of liver, spleen or lymph glands
66	G22	24	0.0746	0.225	G23 0.0287 G22 0.0125	3.5	11	8	2/20/25 2,340 5/19/25 2,280	Living and apparently in good condition 93 days after infection
67	Infect. control	—	—	—	—	—	—	—	1,640 1,930	Still living
64	Infect. control	—	—	—	—	—	—	—	2,530 1,700	Died 5/20/25; generalized tuberculosis

* All injections of compounds made intravenously. On March 17, 1925, Rabbits 65 and 66, receiving G22, showed no improvement over the controls, so treatment with G22 was discontinued and G23 substituted. No improvement was noted in any animal until approximately the fifty-fifth day of treatment, when a slight difference was noted, such as less induration, the lesion taking on more or less the character of an ulcer. On the sixty-third day of treatment there was a marked difference between the treated and the untreated animals.

† Infected intradermally, Feb. 19, 1925. Infected in six places, 0.2 c.c. of a suspension of B. tuberculosis bovine (0.001 gm. of bacilli to each cubic centimeter).

‡ G23 was a gold-protein compound.

§ G22 was composed of gold and sodium thiosulphate.

became inflammatory and raised. Four of the rabbits were then given intravenous injections of sodium-gold arsphenamin (prepared by Dr. George W. Raiziss for us) in the dose of 5, 6, 7 and 8 mg. per kilogram. Eighteen injections in all were given.

The infected but untreated control animal died of visceral tuberculosis in one hundred and twenty days. Two of the treated animals died about the same time of tuberculosis. One of the treated rabbits survived, with a gain of weight of

320 gm., and lived for one hundred and sixty days, when it died. At necropsy generalized tuberculosis was found.

The remaining treated rabbit was living at the end of one hundred and sixty-three days, the weight having increased 150 gm. The skin was entirely healed.

Combined Scarification and Intradermal Inoculations in Rabbits.—In the following experiment, two rabbits were inoculated by scarification and the rubbing in

TABLE 4.—*Determination of the Curative Properties of Gold Arsphenamin in Experimental Tuberculosis of the Skin in Rabbits* *

Rabbit†	Treated with Sodium Gold Arsphenamin	Number of Doses of Compound 0.010 Gm. per Kg.	Number of Doses of Compound 0.005 Gm. per Kg.	Dose of Each Animal Changed in Accordance with Extent of Skin Lesion, Gm. per Kg.	Number of Doses Under New Treatment Scheme	Interval Between Infection and Treatment, Hours	Total No. of Injections of Compound	Total Amount of Compound Injected, Gm.	Average Interval Between Treatments, Days	
497	12/5/24	3	5	0.008	18	24	26	0.3662	2.6	Apparently normal for 163 days; weight 155 gm. more than at start of experiment; lesions on back healed, no induration; no treatment for 91 days
498	12/5/24	3	5	0.005	18	24	26	0.2592	2.6	Animal living, apparently normal, 160 days; weight 320 gm. more than at start of experiment; lesion on back healed; no treatment for 91 days
499	12/5/24	3	5	0.007	18	24	26	0.3527	2.5	5/20/25; animal died after 120 days; generalized tuberculosis; lesions in lungs, kidneys and intestines; also all lymph glands of the body; liver and spleen showed no lesions
500	12/5/24	3	5	0.006	18	24	26	0.2656	2.6	Dead, 117 days; animal became paralyzed 10 days before death; tuberculosis general except in liver and spleen; all lymph glands, lungs, kidneys, intestines, omentum, tuberculous peritonitis; spinal fluid showed tubercle bacilli
474	Infected untreated control	—	—	—	—	—	—	—	—	Dead, 120 days; generalized tuberculosis; lesions in all lymph glands, lungs, kidneys, along spinal vertebrae and intestines; no lesions in liver and spleen

* B. tuberculosis, bovine, strain "H" Pennsylvania Bureau of Animal Industry used in experiment.

† Infected Dec. 4, 1924. Area of skin of back and side approximately 5 by 7 cm. scarified by linear markings with sharp scalpel and a suspension of B. tuberculosis, bovine, 0.001 gm. per cubic centimeter rubbed in with sterile swab.

of an emulsion of the bovine tubercle bacillus on one ear, and intradermal inoculation in four or five areas on the other ear. One animal was first treated with G22 (sodium and gold-thiosulphate) and later with G23 (a gold-protein compound). The rabbits lived for two hundred and twenty-two days, at the end of

which time one died and the other was killed. Both exhibited systemic tuberculosis. A marked difference in the appearance of the two was, however, evident, the treated animal showing distinctly less infiltration and ulceration (Fig. 2).

SUMMARY

While we cannot claim to have prevented the generalization of tuberculosis in inoculated guinea-pigs and rabbits by means of injections of gold compounds, we have observed a demonstrable influence on the cutaneous lesions, and in some animals a considerable prolongation of life beyond the average of the infected controls.

We believe that in chemotherapeutic investigations of this kind rabbits are to be preferred to guinea-pigs, and intradermal inoculation or infection by scarification is preferable to subcutaneous inoculation. By the latter methods, the disease runs a slower course, thereby giving the remedies employed greater opportunity.

Guinea-pigs succumb so rapidly to experimental infection with the tubercle bacillus that the conditions are rather different from those which obtain in the human species.

No absolute deductions can be drawn from the experiments here reported. We believe, however, that they indicate that a favorable, though not curative, influence is induced by gold therapy. Taken in connection with the work done abroad, they offer the hope that perhaps with a more ideal gold compound something may be achieved in the treatment of cutaneous or of general tuberculosis.

ULCUS VULVAE ACUTUM (LIPSCHÜTZ) *

REPORT OF A CASE

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CHICAGO

Lipschütz [1] has employed the name "ulcus vulvae acutum" to designate those cases of acute ulceration of the vulva occasioned by the organism which he has named *Bacillus crassus*, an organism apparently identical with Döderlein's bacillus. In that certain other disorders may be responsible for acute ulceration of the vulva, most notably that type produced by Plaut-Vincent's organism, the disease under consideration will be designated as ulcus vulvae acutum (Lipschütz). To Lipschütz and Scherber are due most of the credit for our knowledge of this comparatively newly described genital affection. It is an entity and is recognized as such in Austria and some of its neighboring countries, but apparently no instance of this disorder has as yet been observed in America. Last year a group of five cases was reported by McDonagh [2] of London, the remaining fifty-one reported cases being in the German literature. The latter have been collected and reported recently in monograph form by Lipschütz.

The first case of this disorder was seen by Lipschütz in 1904 and described as ulcus pseudo-venereum. This case, along with four others observed by him, was again reported in 1912 under its present name Since that time Lipschütz has recorded seven more cases, Scherber twenty, Volk four, McDonagh five, and Oppenheim, Kummer, Appel, Brünauer, Dreyer and several others, one each, most of which have been so carefully studied that collectively they afford accurate knowledge of this disease.

Although not transmissible by sexual intercourse or otherwise, ulcus vulvae acutum (Lipschütz) is of social and forensic significance in that clinically it resembles certain venereal disorders.

Having studied the following case at the Allgemeines Krankenhaus in Vienna, through the kindness of Professor Riehl I am permitted here to report it.

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* Read before the section on Dermatology and Syphilology at the Seventy-Sixth Annual Session of the American Medical Association, Atlantic City, May, 1925.

1. Lipschütz, B.: Ulcus Vulvae Acutum, Dermatologische Studien, Leipsig, L. Voss **25,** 1923.

2. McDonagh, J. E. R.: Acute Ulceration of the Vulva, Brit. J. Dermat. **36**:285 (July) 1924.

REPORT OF CASE

History.—K. M., a housemaid, single, aged 29, was admitted to the hospital on May 25, 1924, complaining of burning pain in the genital region, fever, anorexia and a feeling of general indisposition which had been present since the last day of her menstrual period, three days previously. Her first symptom was a severe chill, following which her temperature, taken by herself, was 40.1 C. (104.1 F.). She averred that she had never had sexual relations. She gave no history of tuberculosis, and there was no other noteworthy personal or family history.

Examination.—On admittance to the hospital, her temperature was 38.2 C. (100.7 F.), and her tongue was heavily coated. On the inner surface of each small labium was an ulcer, almost dime sized, approximately round, and with an abruptly sloping margin. An erythematous areola, 2 to 3 mm. in width, surrounded each ulcer. Their bases were fairly soft and covered with an adherent fibrinopurulent exudate. There was also a small pea-sized ulcer of the left labium bordering the hymen, similar in all essentials to the other two ulcers, but deeper. The hymen, slightly lacerated in several places, was approximately intact. The inguinal glands were not palpable. A smear preparation from each ulcer showed numerous, thick, short and long, gram-positive bacilli. Dark-field examination revealed no spirochetes, and the Wassermann and Meinicke reactions were negative. Cultures made at this time on ascitic fluid agar showed later only a staphylococcus growth. An attempt at auto-inoculation, made at the inferior border of the left large labium, proved a failure. Histologic sections stained with pyronin methyl green showed no Unna-Ducrey streptobacilli but a few bacilli similar in structure to those seen in the smear preparation.

Course.—Three days later the fibrinous deposit of each ulcer became partially detached, and on the following day it became completely detached, exposing a red, finely granular floor. At this time the body temperature became normal, and so remained. Within the following week the ulcers almost entirely healed, and the patient, having developed no new lesions, was dismissed. Treatment, aside from the employment of simple antiseptics locally, was symptomatic.

Histology.—Hematoxylin-eosin sections showed a sharply marginated ulcer with a thick necrotic floor. A dense infiltrate extended into the subcutaneous tissue and laterally into the edematous corium well beyond the margin of the ulcer. Beyond the dense infiltrate were ramifications of perivascular infiltration. The cellular infiltrate in the vicinity of the surface of the ulcer consisted largely of polymorphonuclear leukocytes; more deeply it was composed, for the most part, of lymphocytes. Fresh hemorrhage was marked throughout the entire portion of the corium which lay beneath the surface of the ulcer. The blood vessels were engorged, and their coats were swollen and in places permeated by leukocytes.

SYMPTOMATOLOGY

In ulcus vulvae acutum (Lipschütz), ulcers appear suddenly on the mucous membrane or the skin of the vulva or its vicinity, which are of two clinical types. The more formidable variety is spoken of as the *gangrenous* type and is the less common. The lesions are multiple, few in number, and are most common on the medial surface of the

labia minora. Burning sensation of the outer genitalia is marked, and the vulva may be red and swollen. These ulcers develop suddenly, and the patient has a rise in temperature from 38 to 40 C. following a chill; they are pea to dime size from their onset and become deeper within forty-eight hours, although the diameter increases only slightly. The ulcers are usually round or oval and have a red areola and rather steeply sloping, soft and sometimes undermined margins. Their bases, almost from the onset, are covered with a firmly adherent, thick or thin, grayish-yellow to bluish-black membrane which begins to loosen from the margin on the second or third day, usually becoming completely detached within the following twenty-four hours, exposing a soft, smooth-floored ulcer with a thin grayish fibrinous or fibrinopurulent

Fig. 1.—*Ulcus Vulvae Acutum* (Lipschütz), with three ulcers of labia minora.

coating. Fever and pain disappear at this time. This second membrane comes off within from three to five days more, exposing a bright red base which heals within the following week or ten days, resulting in sharply circumscribed, smooth, atrophic scarring. The more common, or *venereal* type, so-called because of its striking resemblance to chancroid, usually occurs in the absence of constitutional symptoms and with but mild subjective symptoms. These ulcers vary greatly in number and size, are usually more shallow than those of the former type, and have sharply defined, often serrated, more or less steeply sloping undermined borders. They are most common in the vicinity of the vaginal introitus. They may be round, oval, irregular or even fissure-like, and their bases have a fibrinopurulent coating, and are

sensitive to touch. This is the subacute form, often persisting for a month or more, new lesions appearing as the older ones heal. Occasionally associated with this type, but never occurring independently, is what Lipschütz describes as the *miliary* form, in which few or numerous, pinhead-size or slightly larger ulcers of acute onset, with slightly elevated erythematous margin and slightly depressed pus covered center, appear on the labia majora and less frequently on the small labia or perineum. They are sensitive to touch and disappear within a few days.

OTHER SYMPTOMS AND EXCEPTIONS

What kind of a primary lesion precedes the ulcer formation is unsettled, as a developing lesion has not been accurately observed. Whatever it is, it rapidly breaks down into an ulcer. It seems most logical that it should be either an inflammatory soft nodule or an erosion, more likely the former. McDonagh states that the initial lesion seems to be an ulcer.

The lesions not uncommonly appear immediately following the menstrual period, but they occur also prior to the first menstruation. One patient had war amenorrhea and another had been hysterectomized. A large number of the patients were sufferers of dysmenorrhea.

Subjectively, itching and particularly burning, of the outer genitals is present, sometimes extending into the vagina. A burning sensation on urination sometimes occurs. Pain may be so severe as to prevent walking, but this is not the rule. The ulcers remain very tender until their floors become clean, which is usually in less than a week.

Less than half of the cases are preceded by a chill, following which the body temperature rises to 39 or 40 C. The fever is accompanied by headache and malaise and lasts for from two to five days, at which time it becomes normal and so remains. In cases in which the temperature did not become normal within this period of time, pulmonary tuberculosis, enteric fever and other accompanying infections have been demonstrated.

The lesions are multiple and with a marked tendency to asymmetry. One case is on record which consisted of but a single lesion. When the lesions are large they are usually few in number, and when small, numerous. In rare instances large lesions are followed by numerous small ones. Ordinarily, the ulcers remain discrete, but occasionally they fuse. In one case the vaginal introitus became a single annular ulcer. The lesions have a stale but not a putrid odor.

Sites less frequently involved than the medial surface of the small labia and the vaginal introitus are the large labia and perineum elsewhere, the genitocrural folds and the upper medial surface of the thighs. In Brünauer's case, the lesions were altogether perigenital. In one of Volk's cases, there was an ulcer of the cervix of the uterus.

Vulvitis and a vaginal discharge frequently accompany the disorder. There is usually no inguinal adenopathy, although occasionally lentil-sized glands occur. Gross' patient, with a tender "doughy" gland, the size of a pigeon's egg, was an exception. Anuria was present in two cases.

The disorder runs a self-limited course, the ulcers healing within from ten to thirty days with the formation of smooth, sharply circumscribed atrophic scars. Relapse has been noted in a small percentage of cases. Gross' patient had five attacks in four years; one of Lipschütz's, seven attacks in nine years. It has never been seen in patients with demonstrable syphilis, but it occurred during a gonorrheal infection in one of McDonagh's cases.

Fig. 2.—*Bacillus crassus*, photomicrograph of smear preparation from ulcers illustrated in Figure 1. The organisms occur singly or in small groups, are large, of unequal length but of equal width, and have right-angled ends.

ANALOGOUS CASES IN MALES

In 1914, Lipschütz reported the case of a man with ulcers about the anus, and later, Volk, a case with miliary ulcers at the root of the penis, the perineum and about the anus, both of which showed organisms morphologically identical with those of ulcus vulvae acutum in smear preparations, and they contained no spirochetes or Ducrey bacilli. Neither case was culturally verified.

ETIOLOGY, BACTERIOLOGY AND PATHOGENESIS

Ulcus vulvae acutum (Lipschütz) has a predilection for virgins intactae, 70 per cent. of the patients belonging to this group. Of those who were not virgins, it has occurred more frequently in married women. It has been reported in females from 3 to 38 years old, and is most common between the ages of 14 and 20. Usually, those afflicted are otherwise apparently very healthy, aside from the fact that many give a history of dysmenorrhea. Scherber reported ulcus vulvae acutum in two sisters who slept together.

Pus from the ulcer shows almost exclusively ever-present, exceedingly numerous, gram-positive, thick and unusually long bacilli. These organisms are of constant width, 0.5 microns, but vary from 3 to 9 microns in length. They are usually straight, with right-angled ends, and solitarily arranged, short chains being exceptional. They are nonmotile, stain uniformly, and are most numerous in new ulcers. Neisser's stain demonstrates an occasional vacuolated organism. A few of these bacilli are sometimes seen within leukocytes. This organism has been named *Bacillus crassus* by Lipschütz, although extensive study by Scherber and others has proved its identity with Döderlein's bacillus, a constantly present saprophyte of the vagina and external genitalia. Structurally, it is similar to *B. anthracis, B. subtilis* and other large bacilli.

Bacillus crassus has been demonstrated in increased numbers in vulvitis of children, urethritis (especially in prostitutes), cervicitis, acute purulent salpingitis and other disorders of the female genito-urinary tract in the absence of gonococci, tubercle bacilli and other organisms. Organisms identical with it have been demonstrated in the mouth and in pulmonary abscess, along with Vincent's organism. *Bacillus crassus* has frequently been demonstrated in chancre, chancroid and tuberculous ulcers, but apparently does not alter the clinical characteristics of these disorders. It is evident, however, that it may be widely distributed as a saprophyte. On the other hand, although lesions of ulcus vulvae acutum (Lipschütz) frequently harbor such organisms as "diphtheroids" and staphylococci, Vincent's organism, although a saprophyte, is rarely found in them, which makes it appear that there is some antagonism between them.

A great deal of cultural and other experimental work with this organism has been performed by Scherber, E. Lowi, Lipschütz and others. Aerobic cultivation on ordinary mediums was a failure. Scherber first cultivated it in pure culture in a nitrogen anaerobic atmosphere. Lowi found the organism facultatively anaerobic but definitely not with predilection for anaerobic conditions. Lipschütz considers aerobic cultivation of this organism best because of its simplicity,

although the growth is more luxuriant anaerobically. He found serum-sugar-agar stab cultures and particularly maltose-ascitic-fluid-agar plates most serviceable. Pus transferred in parallel columns onto ascitic-fluid-agar plates shows an abundant growth of staphylococcic, pseudodiphtheric and other micro-organisms within twenty-four hours. At the borders of this growth are delicate, scarcely visible, hairy outrunners, which are composed entirely of colonies of *Bacillus crassus*. This material, microscopically verified, when transferred to another ascitic agar plate shows *Bacillus crassus* in pure culture within twenty-four hours at 37 C. (98.6 F.). When fully developed the growth is

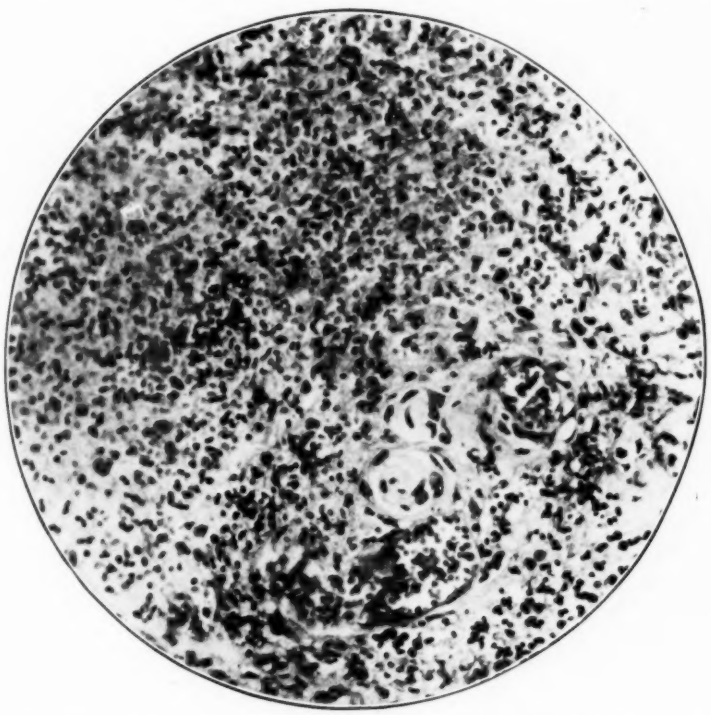

Fig. 3.—Photomicrograph of tissue well beneath base of ulcer (× 230), showing marked hemorrhage into lowermost corium, polymorphonuclear and lymphocytic infiltration, and engorged and edematous blood vessels.

grayish yellow and of "Haarskulptur" appearance. In young colonies the bacilli are found solitarily or in small groups, as in the smear preparations, but in older colonies they are present chiefly in long threads. Loeffler's serum may be used for further cultivation, but old Loeffler cultures show markedly teratologic forms.

Almost every conceivable method of inoculation of pure cultures, as well as of pus from ulcers, has been employed on man and animals.

and, with the exception of one of Scherber's experiments, altogether with negative results. He succeeded in producing genital ulcers in two rabbits by intracutaneous injection of their external genitalia with a young pure culture of *Bacillus crassus* suspended in physiologic sodium chlorid. Flat, round, shallow ulcers, the size of a hemp seed, with a yellow necrotic coating appeared on the day following the injection. These healed within three days. From these ulcers Scherber recovered the organisms and grew them in pure culture. Although technically Koch's postulates are fulfilled, infection of the human female genitalia with pus from the ulcers and with pure cultures of this organism have been uniformly negative. The disorder is best explained, therefore, as an autoinfection, a saprophytic organism becoming pathogenic to an individual because of factors which might lower the local or general tissue resistance. Menstruation is one of these numerous factors. This appears logical in view of the fact that the body is constantly harboring pneumococci, streptococci, Vincent's organisms, etc., as saprophytes, these becoming pathogenic by circumstances which increase their virulence or decrease that of their host.

Histologically, ulcus vulvae acutum (Lipschütz) is characterized by a deep ulcer with undermined margin, and necrosis extending far into or even through the corium. The cellular infiltrate is dense and is composed in its upper half chiefly of polymorphonuclear leukocytes, but in its lower half chiefly of small mononuclear cells. Hemorrhage is usually marked through that portion of the corium lying beneath the surface of the ulcer, erythrocytes frequently being in greater numbers than the other infiltrating cells. The blood vessels and lymph spaces are dilated, and the former are engorged and have edematous, though not proliferated, coats. This swelling is most evident in the endothelium of the intima. Diapedesis is seen here and there in the larger blood vessels. Leukocytes have been reported as being so numerous in the dilated vessels beneath the ulcer surface as to appear as miliary pseudo-abscesses. *Bacillus crassus* is readily demonstrable in the most superficial parts of sections of tissue stained by the Gram-Pappenheim and Giemsa methods.

DIAGNOSIS

Ulcus vulvae acutum (Lipschütz), in view of the characteristic clinical history, clinical appearance of the lesions and constant bacteriologic findings, should cause no difficulty in diagnosis. If the exudate of the ulcer does not show *Bacillus crassus* in abundance in smear preparation, it is not ulcus vulvae acutum (Lipschütz). However, it is possible for these organisms to be present in genital ulcers of other varieties; therefore, the clinical appearance of the lesions and the clinical history must be given more than usual weight.

This disorder must always be considered in cases of ulcers of the female genitalia with intact or partially intact hymen, as well as in others.

Clinically, the greatest similarity to ulcus vulvae acutum (Lipschütz) is encountered in chancroid and in acute ulceration of the vulva occasioned by Vincent's organism. In chancroid, a history of recent exposure is of some value, and the lesions, although tender, usually multiple, soft ulcers, are commonly associated with regional adenopathy, with less acute onset and more chronic course, and are auto-inoculable. Ducrey's streptobacillus, the etiologic organism, although less easily demonstrated, is usually in chain form, and is gram-negative, short, and often has bipolar staining. Acute ulceration of the vulva caused by Vincent's organism, a symbiosis of *B. fusiformis* with *Spirochaetae refringens,* is usually accompanied by more severe constitutional symptoms, causes greater destruction of tissue and runs a more chronic course. The exudate of the ulcers has a putrid odor in contrast to the stale odor of ulcus vulvae acutum (Lipschütz), and the causative organisms are easily demonstrated in smear preparations.

Other disorders which should be borne in mind are atypical chancre and herpetic ulcers, as well as such rare conditions as tuberculous, gonorrheal, diphtheric and other ulcers. It is probable that some of the cases described by Newman as "aphthae of the female genitalia" and by Welander as "superficial anogenital ulcers in women" were ulcus vulvae acutum (Lipschütz).

SUMMARY

In ulcus vulvae acutum (Lipschütz) we have to deal with a comparatively newly described nonvenereal genital infection of acute onset and self-limited course. The ulcers occur in "gangrenous" and "venereal" types. In the former, fever and other constitutional symptoms occur at the onset of the disorder. The lesions in this type are few in number, varying in size from that of a pea to that of a coin, tender, deep, soft, with fibrinopurulent coating, red areola and with predilection for the inner surface of the small labia. They heal in about two weeks, with scar formation. In the venereal type there are usually no constitutional symptoms, the lesions are usually smaller, shallower, more numerous, most common about the vaginal introitus, and the course is more chronic. Various factors capable of lowering the patient's local tissue resistance apparently render the saprophytic *Bacillus crassus* pathogenic. This organism is apparently identical with Döderlein's vaginal bacillus and is ever-present in the lesions, existing as a thick (0.5 micron) and usually long (3 to 9 microns), gram-positive bacillus, with ends sharply right-angled to its long axis. It is easily demonstrated in smear preparations from the ulcer surface and

can be cultivated with but little difficulty, aerobically and anaerobically, on maltose-ascitic-fluid agar and sugar-serum-agar. This disease occurs chiefly in persons between the ages of 14 and 20 years, and is more common in virgins than in others. With the exception of the consitutional symptoms often present at the onset, it is purely of local nature. It sometimes recurs. It is well defined, clinically and bacteriologically, and apparently is an entity. Only in the instances of two of Scherber's experiments on rabbits has the disorder been reproduced. From these lesions he recovered the organisms in pure culture. It has not been reproduced in human beings. Ulcus vulvae acutum (Lipschütz) resembles most closely chancroid and acute ulceration of the vulva caused by Vincent's organism. About sixty cases are on record. Its rarity cannot be determined at this time. It is probable that we occasionally observe this disease and mistake the diagnosis. A case is here reported with moulage and photographic illustrations.

25 East Washington Street.

DISCUSSION

DR. JOHN BUTLER, Minneapolis: Dr. Finnerud has given us a very valuable contribution in this paper. The fact that clinically the lesions are both gangrenous and erosive might easily lead one to mistake it for a gangrenous or erosive balanitis, but of course in both of these Vincent's organism is easily detected. In looking at the moulage, which shows the clear-cut ulcer with the yellow white base and surrounding inflammatory areola, it suggested to me an aphthous ulcer, and I wonder whether the aphthous ulcer can be clearly differentiated from the condition described by Dr. Finnerud.

DR. JEFFREY C. MICHAEL, Houston, Texas: Dr. Finnerud's case recalls one reported by Olson in 1920 in the ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY under the designation of veneroid ulcer. This is synonymous with the disorder reported by Welander who, I believe, antedated Lipschütz.

Some time after Olson's case was recorded, I saw this condition in a nurse who had been a widow for four years. Other infections were rigorously excluded. In the single ulcer present, *Bacillus crassus* was found in abundance. The ulcer disappeared in a month under alcohol applications.

Does Dr. Finnerud intend to include Welander's ulcer under that of Lipschütz?

DR. FRED WISE, New York: It is easy to understand why diseases like Madura foot or creeping eruption are not seen except in certain geographic localities, but it is remarkable that cases such as Dr. Finnerud described should not be encountered more frequently in large cities like New York. Judging from the literature, I think we are sometimes likely to pass by such cases as being Vincent's infection or some other more obscure disease and fail to perform a sufficient number of bacteriologic examinations in an attempt to identify the disease. The picture shown by the essayist on the screen depicting the rod shaped bacillus might easily fail of recognition by any but experienced bacteriologists. These lesions usually have so many different organisms in the smear that unless one is an expert bacteriologist he is likely to pass them over and not recognize the causative organism.

Another point is, do these lesions occur on other mucous membranes, especially in the mouth? About once a month we encounter a lesion in the mouth which we know is not pemphigus, which we know is not Vincent's infection, and which we know is not syphilis, and there are many other conditions that we can exclude, and the question arises whether they have any connection with this particular type of lesion. I see no reason why the bacteria infecting the vulva might not infect the mucosa of the mouth as well.

Dr. M. B. Parounagian, New York: Of the many women treated in our clinic at Bellevue I have never seen any with a condition that sounds like the description given by Dr. Finnerud. We see chancres and occasionally chancroids, but never anything like this. I will watch for it, but probably it does not occur in New York.

Dr. Paul A. O'Leary, Rochester, Minn.: We have studied two of these cases, one of which several of the men present have seen. One of the patients was an unmarried woman, aged 30, and the other a child, aged 9 years. I wish Dr. Finnerud in his closing remarks would emphasize the treatment of ulcus vulvae acutum, as we have been unsuccessful in our efforts thus far to cure either patient.

Dr. Clark W. Finnerud, Chicago: Regarding the treatment, if the disorder does not clear up spontaneously, almost without treatment, I believe that it cannot be ulcus vulvae acutum (Lipschütz). This clears up under simple local antiseptics, and any case that does not respond in that manner is worthy of special mention.

As to its differentiation from Vincent's ulcers, besides the microscopic examination, Vincent's is a more severe infection, is usually associated with more severe constitutional symptoms, and is ordinarily more of a foul-smelling gangrenous process. We do have the superficial or erosive type, however, but even that does not respond so easily to treatment as does ulcus vulvae acutum (Lipschütz).

As regards the aphthous ulcer, it is presumed by the Austrians that most, if not all, of the cases so classified are ulcus vulvae acutum (Lipschütz). The cases in which *Bacillus crassus* can be demonstrated in the lesions are considered ulcus vulvae acutum of Lipschütz. *Bacillus crassus* has been demonstrated in the mouth along with the symbiotic organism of Vincent, both as a saprophyte and in the presence of pathologic conditions. It has also been demonstrated in pulmonary abscess along with Vincent's organism. Two cases are on record of *Bacillus crassus* infection of the anogenital region of the male; one was reported by Lipschütz in 1914, in which he demonstrated in smear preparations from the surface of the ulcer what was presumably this organism. The other case, likewise without cultural verification, was reported later by Volk, in which the patient was a man with miliary ulcers at the root of the penis and about the anus. Why we have not seen the disorder in this country I cannot explain.

MADURA FOOT (MYCETOMA)

THE BLACK GRAIN VARIETY IN A NATIVE AMERICAN *

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HAGOPE MISKDJIAN, M.D.
AND
HARVEY S. THATCHER, M.D.
CLEVELAND

The term Madura foot was first introduced by Colebrook,[1] in 1846, to describe a growth most frequently noted on the feet of natives in Madura, India. The condition, however, had been known for many years and probably had been confused with elephantiasis. In 1860, Vandyke Carter [1] first designated the condition as mycetoma.

Sutton [2] would define the disease as a "chronic infectious disorder due to a species of streptothrix, and characterized by swelling of the affected part, with subsequent disintegration of the subcutaneous structures, and the formation of sinuses which open on the surface of the skin." We might perhaps add to this the fact that these sinuses discharge grains either black, white or red. The white, also called yellow or ochroid type, is the most common, and the red is rare.

OCCURRENCE OF MYCETOMA

The disease is endemic in certain parts of India, and is seen at times in Italy, Africa, the Philippines, South and Central America, Mexico and the United States.

In 1921, Boyd and Crutchfield [1] carefully reviewed and studied the subject of mycetoma in their masterly monograph. They reported especially the cases seen in North America, and were able to collect thirty. One was from Canada, fifteen from the United States, thirteen from Mexico, one from Panama and two from Cuba. In the United States, seven were residents of Texas. There were only three women

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* Read before the Section on Dermatology and Syphilology at the Seventy-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., May, 1925.

* From the Departments of Dermatology and Syphilology and of Pathology of the Western Reserve University and of the Lakeside and Cleveland City Hospital.

1. Boyd, M. F., and Crutchfield, E. D.: A Contribution to the Study of Mycetoma in North America, Am. J. Trop. Med. **1**:215 (July) 1921.

2. Sutton, R. L.: Diseases of the Skin, Ed. 5, St. Louis, C. V. Mosby Company, 1923, p. 1127.

in the list. Nineteen of the patients were native Mexicans, and of these fourteen were laborers accustomed to go barefooted. Since their report, Pagenstecher,[3] has observed three more cases in Texas, and Lovejoy and Hammack [4] have reported four cases from the Mexican quarter of Los Angeles. Two of these cases had already been included in the report of Boyd and Crutchfield, so that we have a total to date of twenty cases reported in the United States. Nineteen of these were of the white, yellow or ochroid variety, and only one of the black grain type. This patient, an Italian woman, was observed and studied by Wright [5] of Boston in 1898. As she had been in this country only six years, it is possible that her infection was contracted in Italy.

A second case of black grain mycetoma is now in the dermatologic service at the Cleveland City Hospital, and it is our privilege to report it here.

REPORT OF A CASE

History.—The patient, a well built Mexican, about 26 years old, was born in Texas. He lived there until he came North about two years ago. He had always enjoyed good health and had worked on farms, where he was in the habit of going barefooted. About five years ago, he noticed a limited swelling in the middle of the plantar surface of the left foot. No history of traumatism was obtained. Only a few months before admission the swelling began to spread and involved also the dorsum of the foot. Small black points were formed on the sole, and the patient soon had the habit of bursting open the epidermis and squeezing out little black grains. The subjective symptoms have always been very mild, and the patient was able to work until admission to the hospital last November.

Physical Examination.—The left foot was considerably swollen. The swelling started in front of the malleoli and extended to the toes. On the dorsal surface there was marked hyperpigmentation of the skin extending backward from the middle of the toes. There were no open sinuses.

On the plantar surface the lesions were limited to the middle portion. The concavity of the arch was replaced by a flat or even convex surface. The tissues were hard and had lost all elasticity, and the induration was made up of numerous confluent deep nodules. The skin was bluish red. Irregularly distributed over the middle portion of the sole were from fifteen to twenty small crusted areas. There were also a certain number of flat, pustular lesions, and through the intact epidermis could be seen small black points surrounded by pus. On breaking a pustule, a typical black grain was obtained along with a few drops of pus. Exploration with a probe lead into a small sinus, more or less deep. The external opening closed up again within twenty-four hours and became covered

3. Pagenstecher, G. A.: Madura Foot more properly called Mycetoma. J. A. M. A. **78**:1363 (May 6) 1922; Third case of Mycetoma, J. A. M. A. **82**:1692 (May 24) 1924.

4. Lovejoy, E. D., and Hammack, R. W.: Mycetoma. Report of Four Cases, Arch. Dermat. & Syph. **11**:71 (Jan.) 1925.

5. Wright, J. H.: A Case of Mycetoma (Madura Foot), Tr. A. Am. Phys. **13**:471, 1898.

with a crust. The sinuses were mostly confined to the middle portion of the plantar surface only a few of them being present on the lateral border of the foot.

Roentgenograms showed no involvement of the bones. The right foot was normal. General physical examination was negative.

The process was treated with rest, the roentgen ray, arsphenamin, etc., with no benefit. In addition, the sinuses were curetted and Menciere's solution was injected, without especial improvement.

A large biopsy was taken from the plantar surface, and union by first intention followed. At the time of the operation, it was noted that there was no involvement of the muscles.

Histologic Pathology.—The majority of the granules were situated in the deeper layers of the corium. They were lobate or uniform, with edges that were fairly regular. The granules consisted of three zones. The middle zone occupied about one-fifth of the structure and was stained deep brown. The central zone was less dense and was light and dark brown. Definite branching threads were

Fig. 1.—Lateral view of foot showing several closed sinuses.

distinguished which suggested mycelia. The dark brown regions had the appearance of pigment which was amorphous and irregular. The outer zone was a small rim which had a pink tinge.

Around the granules there was a thick, dense, edematous region of polymorphonuclear leukocytes. Some of these were undergoing degeneration. Around this area there was loosely formed connective tissue with loops of capillaries. Here giant cells with oval or round nuclei were often present, as well as in close contact with the granules. Most of their nuclei were peripherally arranged, or there was a tendency toward this arrangement, although a few plasma cells, eosinophilic polymorphonuclear leukocytes and large endothelial cells were present. About one granule there were numerous large endothelial cells. In the connective tissue there were often a few minute regions of hemorrhage. The outer portion of the connective tissue was dense.

The subpapillary layer of the skin in one section was greatly hyalinized, and the brown pigment of old hemorrhage was deposited in it. There was a slight

thinning of the rete malpighii over one nodule. Connective tissue proliferation was noted about the sweat glands and the germinal epithelium, with a slight infiltration of small lymphocytes. In the deeper tissue there was more or less hyperplasia of connective tissue around the small blood vessels and around and between the fat cells. Here there was infiltration of small lymphocytes. A few of the regions in the deeper tissue contained hyalinized connective tissue. There was a slight atrophy of some of the sweat glands.

The differences between our case and Wright's case described by Crutchfield are as follows:

1. The granules did not stain with hematoxylin but were brown.

2. There was an outer pink zone on the edge of the granule.

Fig. 2.—Plantar view of foot showing unruptured pustules and closed sinuses.

3. The pigmented material was not regularly distributed.

4. Atrophy of the sweat glands was not a prominent feature, although it did occur.

5. The giant cells were not irregular.

The pathologic findings corresponded to those mentioned by M. Langeron[6] in his monograph on mycetoma.

Summary.—The pathologic picture was that of a granuloma caused by grains, which suggested mycelia.

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6. Langeron, M.: "Mycétomes" in nouveau traité de médecine, Paris, Masson and Cie, 1922, Part 4, pp. 404-429.

SYMPTOMS

The subjective symptoms of mycetoma are almost nil. Usually there is a history that following a trauma to an extremity and generally to a foot, e. g., a thorn bruise to a barefooted person, the process first began. In the course of some weeks, there was a local swelling which

Fig. 3.—Microscopic section of granule located in the corium. Around the granule polymorphonuclear are leukocytes, phagocytic cells and connective tissue. × 580.

continued to grow and gradually to affect the surrounding tissue in the form of a single and later of grouped bluish-red and light red nodules. These collectively in time, if on the foot, give it the peculiar clubbed appearance which is characteristic of Madura foot. Sooner or later these

nodules open, form sinuses and discharge serosanguineous fluid and grains, either black, white or red. On examining the sinuses one finds them to be deep, extending down to or into the muscles, and even involving the bones.

ETIOLOGY

Carter, in the days before bacteriology, classified the disease clinically as of the white and black variety. When later also cases with red grains were found, the red variety was added. This classification has now been replaced by one based on the nature of the causative organism. This is because we know now that any one of these varieties may be produced by numerous widely differing fungi. With Chalmers and Archibald [7] we would now divide the mycetomas into actinomycoses and maduromycoses.

The actinomycoses include those in which the grains are composed of thin nonseptate filaments without chlamydospores. They may be produced by thirteen different species belonging to the genus *Actinomyces* (Brumpt [8]).

The maduromycoses—the true mycetomas of Pinoy [9] are characterized by grains formed by thick septate mycelial filaments with definite walls and frequently forming chlamydospores. They may be subdivided according to the color of the granules they produce. Nineteen different organisms have been described as causing maduromycosis, belonging to the genera *Madurella, Indiella, Glenospora, Scedosporium, Allescheria, Aspergillus, Sterigmatocystis* and *Penicillium.*

In our own case we have gone to great length to differentiate the organism causing the disease. The fact that at all times unruptured pustules were present made it easy to obtain the granules under aseptic conditions. They were found under the skin embedded in a "characteristic viscid, oily, semipurulent, bloodstreaked fluid." They have a diameter of about 1 mm., and frequently ten or twenty may be clotted together and form a conglomerate mass of about 3 to 5 mm. in diameter. Their color is sometimes smoke gray but usually deep black.

Soaking these granules in 10 per cent. sodium hydroxid for from two to seven days and crushing them between two slides shows on microscopic examination septate mycelial filaments of different sizes and ovoid or spherical brown swellings. Some of these may reach a

7. Chalmers, A. J., and Archibald, R. G.: A Sudanese Maduromycosis, Ann. Trop. M. & Parasitol. **10**:169 (Sept.) 1916.

8. Brumpt, E.: Précis de parasitologie, Éd. 3, Paris, Masson and Cie, 1922.

9. Pinoy, E.: Actinomycoses et Mycétomes, Bull. de l'Inst. Pasteur. **11**:929 (Nov. 15) 1913; **11**:977 (Nov. 30) 1913.

diameter as great as 27 microns. They are interpreted as chlamydospores. Amorphous masses impregnated with brown pigment form the greater part of the granule.

CULTURAL CHARACTERISTICS

Since Nov. 27, 1924, on at least ten different occasions granules were removed and planted on various culture mediums, and always the same growth resulted.

The most favorable medium is ordinary glucose agar. It develops faster incubated at 37 C. than at room temperature. The growth appears within from four to five days as a tuft of delicate white filaments

Fig. 4.—Mycelia and chlamydospores in crushed granules treated with 10 per cent. sodium hydroxid. (Drawing by Miss T. Bergsland.)

springing from the black grain and spreading over the surface of the culture medium. The character of the growth seems to depend a great deal on the humidity of the medium. These cultures grow for about two to three weeks, and may have a diameter of 1½ to 2 inches (3.8 to 5 cm.). Frequently in old cultures, especially on Petri dishes, three different zones may be distinguished: the center duveteuse and brown, a ring of wartlike gray or brown efflorescences, and outside of it gray or white duveteuse growth. The most striking feature is the formation of brown pigment on carbohydrate mediums. About the

fifth or seventh day the carbohydrate agar takes a brownish color around and beneath the growth that deepens and spreads more and more, until finally the medium is stained chestnut brown. Black granules about 1 mm. in diameter appear beneath the surface of the mediums.

Hanging drop cultures with Sabouraud's original glucose peptone broth permit the study of the process of reproduction. There are seen radiating from the center thick, branching septate filaments that become brown when old. Some of the mycelial segments swell up and transform into intercalary or terminal chlamydospores. In some cultures the

Fig. 5.—Typical culture of Madurella americana on ordinary glucose agar; three weeks' growth.

mycelia transform into oidia, and usually one interseptal segment forms two oidia of the diameter of the mycelium from which they rise. Also a true conidial apparatus was observed.

The organism stains readily with anilin dyes and is gram-negative.

The clinical, cultural and microscopic characteristics permit the conclusion that we are dealing with a species belonging to the genus *Madurella brumpti*, 1905. However, we cannot identify it with any species so far described, and therefore propose provisionally the name

"Madurella americana." Our findings have been confirmed and concurred in by Prof. E. Brumpt and Dr. M. Langeron of the University of Paris.

ANIMAL EXPERIMENTS

In the past numerous animal experiments have been performed in the hope of better studying the mode of infection, transmission, etc. Most of these have been negative, however.

Musgrave and Clegg,[10] using the ochroid type, inoculated forty monkeys, rabbits, dogs and pigeons. In nearly all the inoculations their results were fruitless, but with three monkeys they were successful. Typical examples of Madura foot developed locally on the feet after

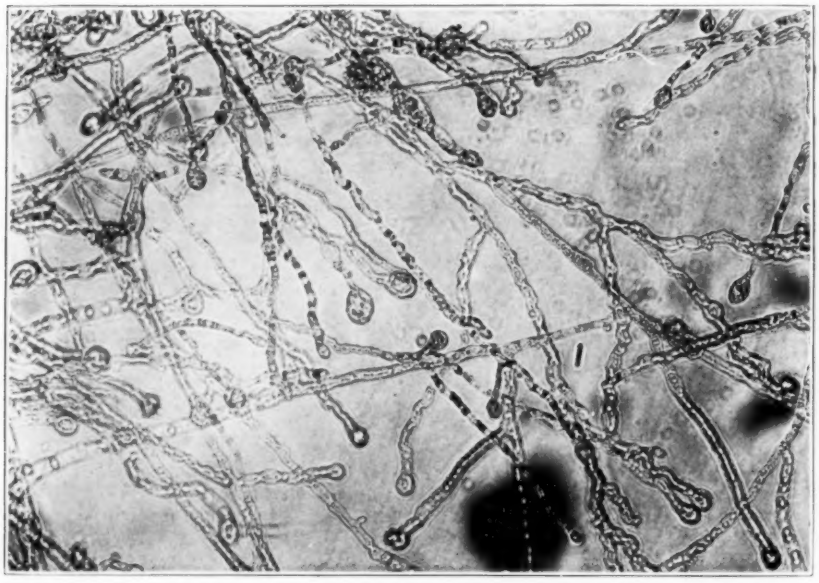

Fig. 6.—Hanging drop culture showing mycelia with terminal and intercalary chlamydospores. × 290.

the injections. The causative organism was first considered as a new species and named *Streptothrix freeri,* but two years later the authors reported its identity with *Actinomyces asteroides-eppingeri* (Pinoy[9]). According to Brumpt, C. Nicolle and Pinoy in 1908 reproduced Madura foot in pigeons with *Madurella tozeuri,* and Yazbek, of Brazil,[11] in 1920,

10. Musgrave, W. E., and Clegg, M. T.: The Etiology of Mycetoma: Report of a Case of the Ochroid Variety Occurring in the Philippine Islands and Caused by a New Species of Streptothrix (Streptothrix freeri), Philippine J. Sc. (Sec. B) **2**:477, 1907.

11. Yazbek, A. K.: Dos Mycetomas, These Inaugural, São Paulo, 1920; Secção de Obras do "Estado de São Paulo," p. 130.

injected cultures of *Madurella mycetomi* intraperitoneally into rabbits. A specific peritonitis resulted, and retrocultures with black granules found at necropsy revealed the original organism.

We likewise have made thirty-five inoculations usually using 1 c.c. suspension of the cultures.

Animal Experiments

Animal	Mode of Injection	Date	Killed After
Rabbit 15	Intra-arterially	1/16	91 days
Rabbit 20	Intraperitoneally	1/16	69 days
Rabbit 170	Intravenously	1/16	Living
Rabbit 160	Intraperitoneally	1/16	Living
Rabbit 12	Intraperitoneally	1/29	Living
Rabbit 150	Intraperitoneally	1/29	Living
Rabbit 66*	Scratches on cornea	2/ 5	7 days
Rabbit 50	Subcutaneously	2/ 5	Living
Rabbit 60	Subcutaneously	2/ 5	70 days
Rabbit 52†	Anterior chamber of eye	2/ 5	Living
Mouse 1	Intraperitoneally	1/21	64 days
Mouse 2	Intraperitoneally	1/21	86 days
Mouse 3	Intraperitoneally	1/21	Living
Mouse 4	Intraperitoneally	1/21	Living
Guinea-pig 90	Intraperitoneally	1/21	86 days
Guinea-pig 10	Intraperitoneally	1/21	Living
Guinea-pig 100	Subcutaneously	1/21	Living
Guinea-pig 40	Subcutaneously	1/21	Living
Guinea-Pig 25	Traumatized leg	1/29	78 days
Guinea-pig 55‡	Traumatized leg	1/29	105 days
5 pigeons	Left foot	2/12	Living
Rat 1	Intraperitoneally	2/20	33 days
Rat 2‡	Intraperitoneally	2/20	7 days
Rat 3	Subcutaneously	2/20	56 days
Rat 4	Subcutaneously	2/20	Living
Rat 5	Subcutaneously	3/ 7	7 days
Rat 6	Subcutaneously	3/ 7	Living
Rat 7	Subcutaneously	3/ 7	Living
Rat 8	Subcutaneously	3/ 7	Living
Monkey 1§	Palma manus	3/ 3	19 days
Monkey 2	Palma manus	3/ 3	Living

* Technic as for Paul's test (Paul, G.: Wien. klin. Wchnschr. **29**:996 [Aug. 3] 1916) for diagnosis of smallpox: The epithelium of the cornea was scratched with a fine needle and suspension of Madurella americana brought into the conjunctival sac. The animal was killed after seven days, and the enucleated eyes placed in an alcoholic solution of mercuric chlorid. No reaction was noted on examination with the hand lens.

† Small white scar on cornea.

‡ Death caused by intercurrent infection.

§ The monkeys (Macacus rhesus ♀) received injections of a suspension cultures into the palm of the right hand, and injections with suspension of discharge from the abscess of the patient and recently removed crushed granules into the palm of the left hand. Monkey 1 died from pneumonia, but necropsy revealed no evidence of mycetoma infection either at local or general sites of injection.

Rats 3, 4, 5, 6, 7 developed small subcutaneous abscesses that opened after one week and healed within the next three days. Rat 5 was killed seven days after injection and on microscopic examination showed an acute inflammatory process but no mycelia.

All animals that were killed were necropsied and microscopic sections were made of all organs which had a suspicion of any pathologic change. Everything was normal. Twenty-one animals are still under observation and apparently well.

In the past (Noc and Jouenne[12]) it was noted that infection with the black grain variety was usually of less virulence than with other types. This may perhaps explain our lack of success in animal experiments—at least with the monkeys.

SUMMARY

1. Report is made of an infection with the granuloma maduromycosis (mycetoma) in a native American (Mexican) from Texas. This is the twenty-first case reported from the United States and the eleventh from Texas.

2. The infection was of the black grain variety—the first thus far reported in a native American and the second black grain infection reported on the North American continent.

3. All attempts at animal inoculations were negative—using monkeys (*Macacus rhesus*), rabbits, guinea-pigs, white rats, white mice and pigeons.

4. Cultural experiments thus far carried out lead us to classify the causative organism with the Madurella group (Brumpt), and because of its characteristics as a new species which we would term *Madurella americana*. A detailed description of it will be published later.

DISCUSSION

DR. EARL D. CRUTCHFIELD, Galveston: The authors' research is important from a scientific standpoint as the discovery of organisms that might produce a great many of the more common conditions. The authors have pointed out to us that dermatology is a wide field and that if we follow it in all of its amplifications we must be well trained in bacteriology as well as in inspection of the skin. The organism which has been presented here is, apparently at least, a hitherto undiscovered if not a new organism producing Madura foot. Perhaps the most striking thing that came to me in this connection is that we have a new clinical entity. We now know of nineteen or twenty organisms which produce the same clinical characteristics. The probability is that many of these fungi which have slightly different morphologic and cultural characteristics are capable of producing the same disease. The feet shown in the picture were identical with the cases of Madura foot that I have seen. This disease is probably a little better known in North America than we have been led to believe. I recently had material sent to me from two cases occurring in the western part of Texas, which we have not yet identified but feel certain are of the yellow grain Madura foot type. The

12. Noc, F., and Jouenne: Les mycétomes à grains noirs du Senégal, Ann. de l'Inst. Pasteur **36**:365 (May) 1922.

conception Dr. Gammel has given us that Madura foot is due to a great many different organisms, I think is well to carry with us. I hope the essayist will continue this work. It is worthy of a monograph report, and I hope that some day we shall be able to see some of these things where we can spend an hour or two over them in the scientific exhibit.

Dr. Frederick D. Weidman, Philadelphia: My own experience has been limited to the bacteriologic investigation of the organism which Dr. Gammel has been studying. He kindly sent me cultures, and I can confirm the report he has made, at least up to the point of the determination of the genus of the organism. Regarding the question as to whether it is a new species or not, perhaps I would be inclined to keep my mind open in view of the fact that I am rather skeptical about the fungi, not only in this disease but in many others, for instance, in various ringworm infections of the skin. I have been told that the cultures have been sent to Professor Brunt, and he has said that it is a new species. I think it is rather characteristic of the French to incline to a multiplication of species, but I feel that until we have better methods of examining the cutaneous fungi and determining their species, we should keep our minds open on this question.

Dr. Crutchfield touched on the etiology of this disease, pointing out, as Dr. Gammel indicated, that there are probably upward of fifty different species which might cause the disease. This prompts me to emphasize what I have attempted to emphasize before, namely, that it is extremely difficult to attempt to come to a conclusion as to the duality or multiplicity of the causes of diseases which we do not really understand. This question has come up in connection with the granuloma fungoides, the leukemias and other conditions. Here is another example of a disease which, if we did not know could be caused by many different things, we might be tempted to call a clinical entity, and yet it turns out that a great many known etiologic causes may produce the same sort of clinical picture.

Dr. Gammel has produced the organism many different times; he has introduced a new baby to the section. We welcome it with open arms, and the only question to be settled is the validity of the new species.

Dr. John A. Gammel, Cleveland, Ohio: I wish to emphasize that when I claim that this is a new species I do not base that claim on what I have said in the paper. I omitted to report many details as I did not think them of sufficient interest to you.

About the men in Paris, that they considered it a new species is true. The report is four pages long. They told us why they think it is conidial, producing in only one of 186. I doubt that I can produce it again in a drop culture. I will report on this and give the micrologic description later. At present I think it is well in reading the literature on all these organisms to keep an open mind. I think there are six of them, and it is difficult to determine much from the reports, for all the reports are as brief as mine and mine is as short as I could make it.

XANTHOMA WITH ERYTHEMA TELANGIECTODES AND PARAPSORIASIS*

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The development of xanthomatous nodules on a previously existing erythematous base of such wide extent as in the case herein reported does not appear to have been previously noted, at least no published instance of such a condition could be found in the literature. The term used to designate this apparently new clinical type of cutaneous xanthosis was suggested to me by Dr. Jay Frank Schamberg, who presented this patient before the Philadelphia Dermatological Society in March, 1922. At that time the patient presented lesions "involving both upper eyelids at their edges which in that location consisted of yellowish, pea-sized nodules, differing from ordinary xanthoma planum by their round, nodular, markedly elevated rather than flat character, as well as by the fact that the edges of the eyelids rather than the skin surface proper were involved. Covering the forehead, cheeks, lips and chin was a marked erythema of irregular configuration which extended over the eyelids and gave to the xanthomatous lesions an orange color. On the chin, in the region of the erythema, were small elevations varying in size from that of a pinhead to that of a lentil seed. These were reddish in appearance but on pressure exhibited a distinct yellow color. These appeared to be of the same character as the lesions on the eyelids."

When seen two years later and made the subject of this report, a second condition, not previously observed, was found on the body; new lesions had appeared, and his previous condition had become so marked as to cause considerable disfigurement.

REPORT OF A CASE

History.—A white man, aged 25, of Jewish extraction, with a family and personal history which throws no light on his present condition, was referred to me by Dr. H. W. Ostrum in November, 1924, for the treatment of a persistent redness of and a nodular eruption on the face. As far back as he could remember, his face always had had a reddish, although not persistent, blush. In the summer of 1918 this redness suddenly seemed to become more marked and "fixed." Thereafter the entire facial skin presented a constant mottled reddish appearance. He thought it was sunburn, because at that time he was working in a ship-yard and was constantly exposed to the wind and sun. Some months

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* Read before the Section on Dermatology and Syphilology at the Seventy-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., May, 1925.

* From the Research Institute of Cutaneous Medicine, Philadelphia.

later, small yellowish nodules developed on the edges of the eyelids. Since then, these marginal nodules on the eyelids had increased in size and number. Within the last three years similar variously sized lesions had appeared on the chin just below the center of the lower lip and in the nasogenial furrows, in the groins and around the anus. During this time there likewise developed two large isolated lesions, one at the left angle of the mouth and the other on the point of the chin. About two years ago, he noticed spots developing on his body and upper extremities. He said that they had remained exactly the same since their onset, which was insidious. They caused him no discomfort but became more marked after bathing and after they were rubbed.

Generally speaking, the patient felt well, and the only system to which any untoward symptom was referred was the genito-urinary tract. He said that he micturated frequently, but the total urinary output was increased only slightly.

Physical Examination.—The general immediate impression was that of an undersized young adult who, although not poorly nourished in the sense of undernourishment due to a lack of food, appeared so as a result of malassimilation. Examination of the head, chest and abdomen revealed nothing abnormal, with the exception of the breath which had an unusual apple-cider-like odor, the cause of which was not determined. Special search was made for arcus senilis, but only the suggestion of such a disturbance was found.

The erythematous lesions consisted of a confluent, more or less universal patch on the face and of discrete, more or less oval lesions on the trunk and upper extremities.

The facial erythema was slightly scaly and a mottled pinkish red with a distinct yellowish tint. It covered the forehead, especially just above the eyebrows, the temporal regions, the cheeks, behind the ears, extending down over the inferior maxilla, and was found on the chin. It was particularly marked and most intense about the eyes, around the mouth and behind the ears over the mastoids. That it was not altogether due to active or passive congestion was evidenced on pressure or on stretching the skin. By this maneuver, considerable of the redness disappeared, although it did not leave the pallor one ordinarily finds after expelling the blood from an erythematous area; instead, a distinctly yellowish stain remained. On the other hand, if rubbed, no matter how gently, the redness of this facial erythema became much more marked, the degree varying with the intensity of the rubbing. The application of heat or cold likewise increased it. The skin in the region of this erythema, especially that around the mouth, had a thick "feel" as though the deeper parts were infiltrated with some substance. Behind the right ear, between the lobule and the skin over the mastoid, was a narrow yellow fold which appeared to be of a xanthomatous nature and which, besides, appeared to have had its origin in a preexisting erythema; at least this fold blended intimately with the marked erythema of the skin over the mastoid. The fold itself was not firm and gave no sense of anything but a slight fibrotic change. Clinically it appeared to represent an early xanthomatous lesion just before there is any marked fibrosis. This fold was later removed, together with some of the erythematous skin, for histologic studies.

On both shoulders were numerous pigmented spots which in size and appearance simulated ordinary freckles. Scattered among these were pinkish-red, variously sized, but none larger than a lentil, flat, round lesions. Their red color disappeared almost entirely on pressure, and on dioscopy nothing but a pigmented stain remained.

The lesions on the trunk and upper extremities consisted of round and oval, flat, pinkish-yellow, slightly scaly, noninfiltrated patches which varied in size

from that of a dime to that of a nickel when round and were about half an inch (1.2 cm.) wide and about one and one-half inches (3.8 cm.) long when oval. These lesions, which numbered between twenty and thirty, did not itch, but appeared as distinctly yellow spots when, by stretching the skin, their blood content was expelled. Although they became much fainter on pressure, none of them disappeared completely; the stain which remained varied in color from a

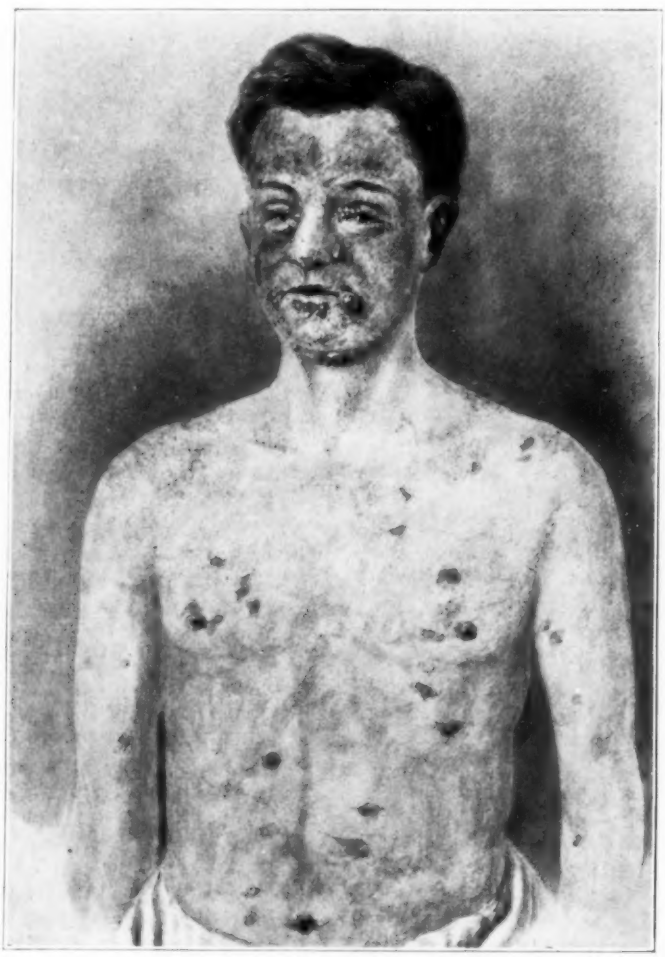


Fig. 1.—Facial erythema and xanthomatous nodules. Note the many small lesions on the chin below the center of the lower lip and in the nasogenial furrows as well as on the edges of the eyelids. The extent of the erythema is apparent.

very faint straw yellow to a deeper yellow. When these patches were rubbed they became more erythematous, but they did not become more turgescent or elevated.

The nodular lesions were located at the margins of the eyelids, the buccal commissures, nasogenial furrows, on the chin just below the center of the lower lip and in the groin and peri-anal regions. The largest one of these nodules, at the left buccal commissure, was about the size of a marble, firm, yellow, not freely movable. At the right commissure was a beginning, well marked, yellow, irregular elevation. In the nasogenial furrows and on the chin were grouped,

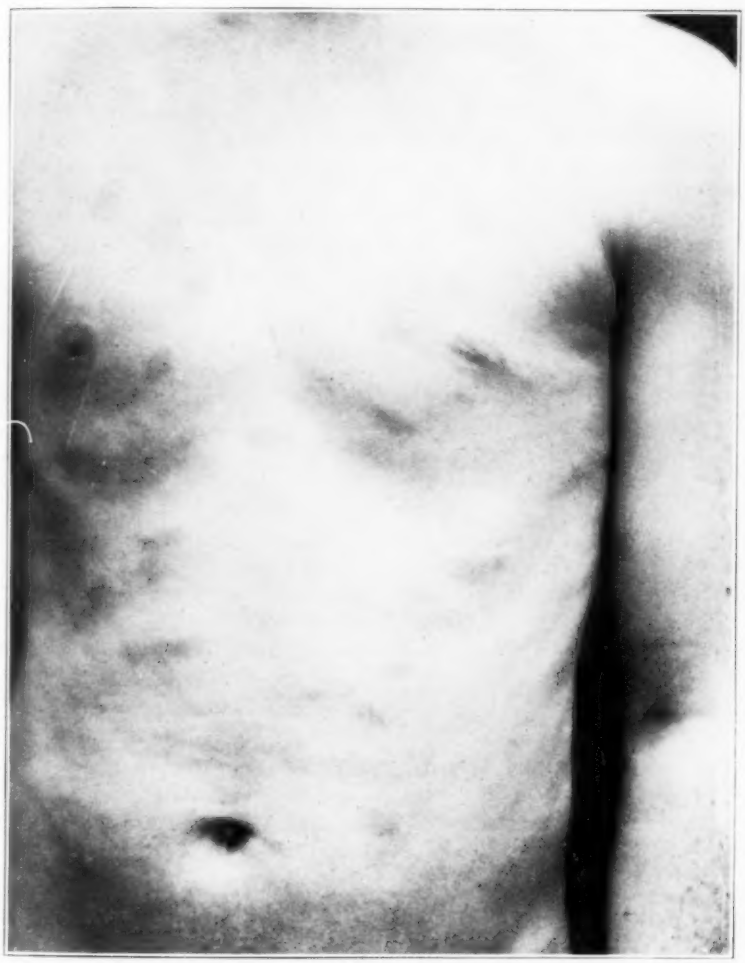


Fig. 2.—Discrete round and oval, pinkish-yellow, slightly scaly lesions on the trunk.

various sized, but none larger than a split-pea, firm, yellow papules. On the chin just below the point, was a large pea-sized, firm, yellow nodule bristling with hairs.

In both groins and around the anal orifice was a combined arrangement of flat, spread out, indurated, yellow masses associated with several elevated nodular

lesions—true xanthomas—differing only in location and size from the lesions seen on the face.

Laboratory Findings.—These included a complete blood count, several urinalyses, several blood chemistry determinations, particularly for the presence or absence of hyperglycemia or hypercholesterolemia, Kolmer and Kahn serologic tests for syphilis and a special urine examination for pentose. The urine examinations were all normal, with the exception that on each of three examinations the specific gravity was found to be extremely low—1.002. The blood cholesterol determinations were made on three different occasions by Mr. Brown (method of Myers), and were found to be within normal limits on each examination. In the other laboratory studies no abnormal findings developed.

Treatment.—The patient was placed on a cholesterol-free diet consisting of roast and grilled meats, fresh vegetables, fruit, skimmed milk and bread. This cholesterol-free diet of Chauffard is probably good therapy for persons with hypercholesterinemia and would appear to be of value purely as a prophylactic in a case such as is reported here. Internally, he was given 15 minims (0.9 c.c.) of oil of turpentine daily. The large xanthomatous nodules were removed by excision; the other smaller ones were destroyed by galvano-cauterization. An

Fig. 3.—Lesions similar to those in figure 2 but located on the upper arm.

attempt was made to modify the facial erythema first by roentgen-ray therapy and later by multiple ignipuncture, with but poor results.

Histopathology.—For this study a nodule from the face and a plaque from the trunk were obtained. For obvious reasons no biopsy could be obtained from the region of the facial erythema, but it was obtained from behind the ear, including the narrow yellow fold and a portion of the erythematous skin over the mastoid process.

The large lesion at the left buccal commissure was removed by circular incision. The clinical diagnosis was definitely confirmed by the histologic structure as found in frozen sections stained with scharlach R and hematoxylin and in other sections fixed and stained in the usual way with hematoxylin and eosin. The frozen sections showed mass deposition of a lipoidal substance, particularly in the derm. The other sections showed a marked connective tissue hyperplasia, a cell infiltrate composed of spindle, polygonal and round connective tissue cells, many of which showed the vacuolated protoplasm of the characteristic foam cell, numerous multinucleated cells, disappearance of the papillae and the interpapillary prolongations and at other points thinning of the epidermis. Between the epidermis and the lipoidal infiltration was the clear noninfiltrated zone observed by many others. In this clear zone no inflammatory reaction was seen.

Clinically, the eruption on the trunk and on the upper extremities was parapsoriasis in plaques. The histologic picture appeared to confirm this. In order to make certain and for further confirmation, the sections were referred to Dr. Sigmund Pollitzer, who spent considerable time in their study before formulating the following opinion: "The histological picture approaches that of an erythrodermie pityriasique en plaques disseminée. There is a decided edema in the subpapillary layer with considerable lymphocytic infiltration. The epidermis shows a moderate amount of parakeratosis. The lesions on the trunk stand in no relation to the lesions on the face, or rather to xanthoma. The patient appears to have two processes totally unrelated. I incline to the diagnosis

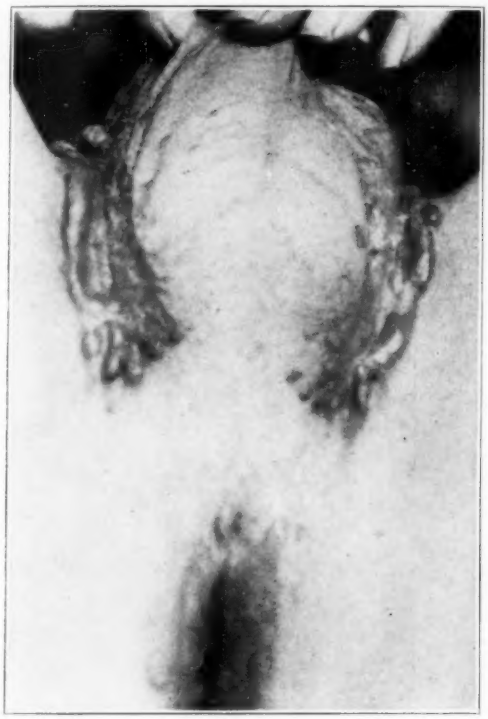


Fig. 4.—Inguinal lesions consisting of flat indurated yellow placards conforming in shape to their location; at the periphery are seen several xanthomatous nodules.

of parapsoriasis." The question of a possible quiescent urticaria pigmentosa was raised, but a section stained with polychrome methylene blue showed no mast cells.

Serial scharlach R and hematoxylin-eosin stained specimens were studied. In the frozen sections the orange-red of a lipoidal infiltration was found throughout. It was found particularly in the papillary and the subpapillary layers, but there was no involvement of the subcutaneous tissue. It was most marked where the epidermis was thinned, the papillae and interpapillary pegs effaced as shown in the paraffin sections. The latter area represents the region of the clinically apparent xanthomatous fold. The lipoidal substance was both intracellular and

extracellular, and was also found in and around the walls of some of the blood vessels in the subpapillary layer. The infiltrate in the subpapillary layer, in the region of the erythema, was more cellular—consisting of round cells, small oval or fusiform cells of connective tissue origin—than the corresponding area in the region of the actual elevation. Although xanthomatous cells were present throughout, the multinucleated ones were mainly found in the latter region, and here definite fibrous tissue formation had definitely taken place. In the papillary

Fig. 5.—Peri-anal lesions. Flattened, yellow, indurated placard completely surrounding the anal orifice; several isolated xanthomatous nodules at periphery of placard.

and subpapillary layers many of the blood vessels were dilated, and they formed as conspicuous a part of the microscopic picture in the region of the erythema as the multinucleated xanthomatous cells did in the region of the actual xanthomatous fold. The fact that the lipoidal accumulations were less marked in the region

of the erythema, in which, however, the cellular proliferation was most marked, appears to confirm the opinion of Weidman[1] that the cellular reaction precedes rather than follows the lipoidal infiltration.

COMMENT

Within recent years, splendid reviews of the pathologic and the probable etiologic factors concerned in the production of xanthosis have appeared in the literature and do not require repetition at this time.

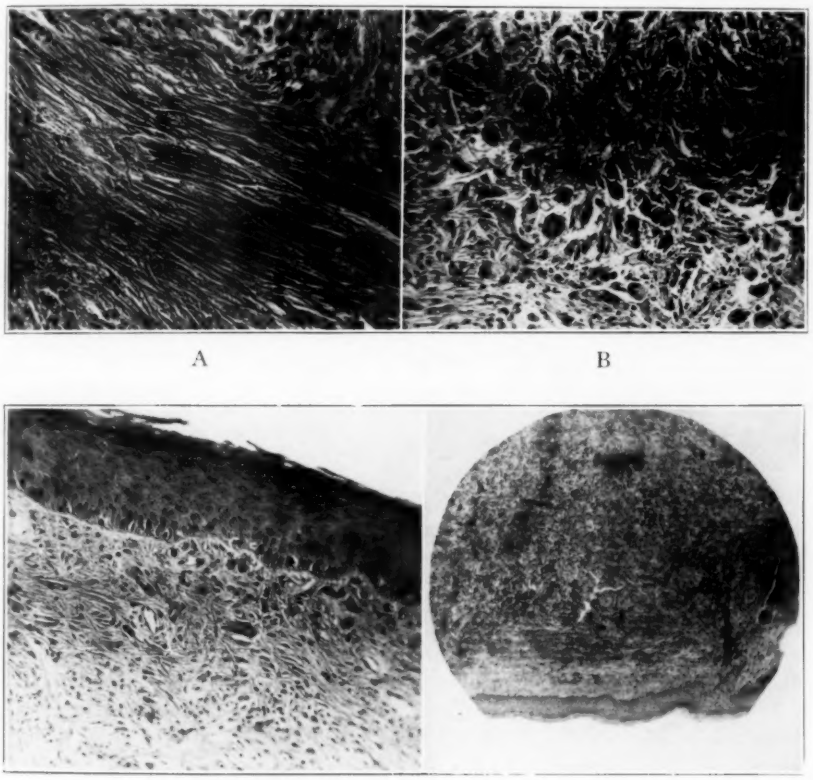


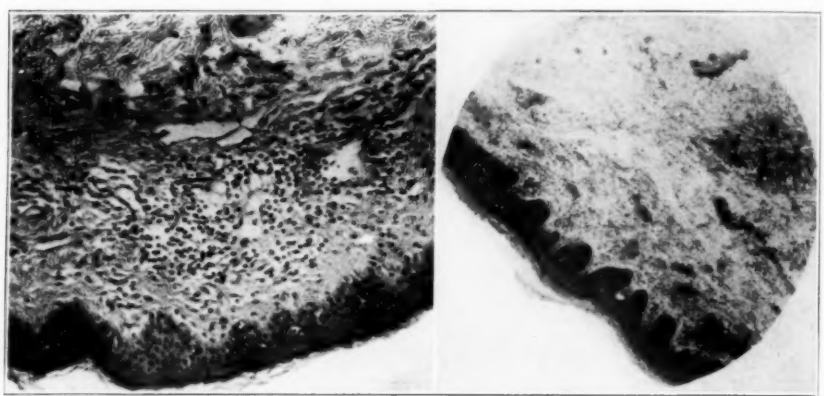
Fig. 6.—Photomicrographs of sections taken from nodule located at left buccal commissure: *A*, high power area showing marked fibrosis; *B*, high power area showing numerous multinucleated cells; *C*, area showing papillary effacement; *D*, low power frozen section showing lipoidal masses in derm and hypoderm with an apparently noninfiltrated area just beneath the epidermis.

This, of course, indicates that clinical reports in themselves are now of little value unless they present features of special interest, such as is found in the present case report.

1. Weidman, F. D., and Freeman, W.: Xanthoma Tuberosum, Arch. Dermat. & Syph. **9**:170 (Feb.) 1924.

Cases of xanthoma tuberosum of unusual clinical interest have been reported by a number of authors, but most of these referred to xanthotic conditions elsewhere than on the skin. Two of these reports, however, deserve special mention because of their bearing on the present case report. In 1913, Macleod [2] reported a case in which the facial lesions consisted, in part, of numerous small yellow, pinhead sized papules situated on a reddish basis of dilated capillaries. On pressure with a diascope, these became paler, the redness was entirely obliterated, and they merely persisted as yellowish macules. The lesions, he states, were instructive in that they pointed to the initial phase being a dilatation of a group of capillaries associated with inflammation and a fatty infiltration.

In 1909, the late Dr. Winfield [3] reported the case of a child whose lesions began as brownish spots which were lentil sized and unaccom-



A *B*

Fig. 7.—Photomicrographs of sections of the plaque removed from trunk: *A*, high power, hematoxylin and eosin stain, shows moderately marked round cell infiltration, dilated lymph spaces with edema in the papillary layer; the epidermis is practically unaffected except for a partial parakeratosis; *B*, same frozen section; no lipoidal masses found.

panied by subjective symptoms. The spots or "stains," as the mother termed them, became more numerous while "small hard lumps" developed on the site of some of the older macules. When first seen, the child presented a generalized eruption of orange-red tubercles and interspersed among these a number of brownish macules. Studies conducted over a period of nearly one year showed that the lesions appeared first as pea-sized, brownish macules, and about one month later as pinhead-sized papules developed on the site of the macule. Exhibited at the Sixth International Dermatological Congress, so that only the back and a por-

2. Macleod, J. M. H.: Brit. J. Dermat. **25**:344, 1913.
3. Winfield, J. M.: J. Cutan. Dis. **27**:112, 1909.

tion of the neck with only the pigmented macules were visible, nearly all of the observers took it for a case of urticaria pigmentosa of the quiescent type, since no evidence of wheal formation was obtained. Besides, however, on histologic examination no mast cells were found. To these two cases our case presents findings that are, to a certain degree, somewhat analogous.

Our patient presents a clinical picture which is extremely striking, namely, xanthoma on a chronic erythematous base, and in addition a clinical picture that is peculiar in the association of two totally unrelated conditions, so far as is known, xanthoma and parapsoriasis in plaques. Aside from the unique association of the latter entity, apparently the first published instance of such a combination, our main interest lies in the facial condition, which appears to be a new clinical form of cutaneous xanthosis.

The plaques on the trunk and upper extremities appear to conform in the highest degree to the condition described by Crocker [4] in 1905 under the term "xantho-erythrodermia perstans." There has been no general acceptance of this condition as an entity distinct from the usual parapsoriasis in plaques. In the latter, it is generally stated that the color is variable, the plaques round or elongated and their surfaces not at all or slightly scaly. Therefore there appears to be no valid reason for establishing a distinction between the two. However, since the etiologic factors concerned in the production of parapsoriasis in plaques is obscure in any case, its assocation with xanthoma is of decided interest from this point of view.

The observation of a patient who presents xanthomatous nodules on a chronic erythematous base—nodules which appear to have sprung from this base—is unique. Histologic studies appear to confirm the clinical impression that the persistent facial erythema represents a part of the xanthomatous process. Although no biopsy could be obtained from the face, serial sections of the lesion obtained from behind the ear, which included a part of the erythematous skin in this region, showed lipoidal deposits and evidences of connective tissue irritation in every section and appeared definitely to point to an etiologic relationship between the xanthomatous nodules and the erythema. As a matter of fact, the supposition that the chronic erythema represents an arrested stage of the irritative reaction caused by cholesterol infiltration—with redness as the clinical sign—is not extraordinary. Pollitzer and Wile [5] and others after them came to the conclusion that the initial changes in xanthoma tuberosum appear to take place about the capillaries of the subpapillary layer and consist in a dilatation of the vessels involved, proliferation of the

4. Crocker, H. Radcliffe: Brit. J. Dermat., April, 1905, p. 119.

5. Pollitzer, S., and Wile, U.: J. Cutan. Dis. **30**:235, 1912.

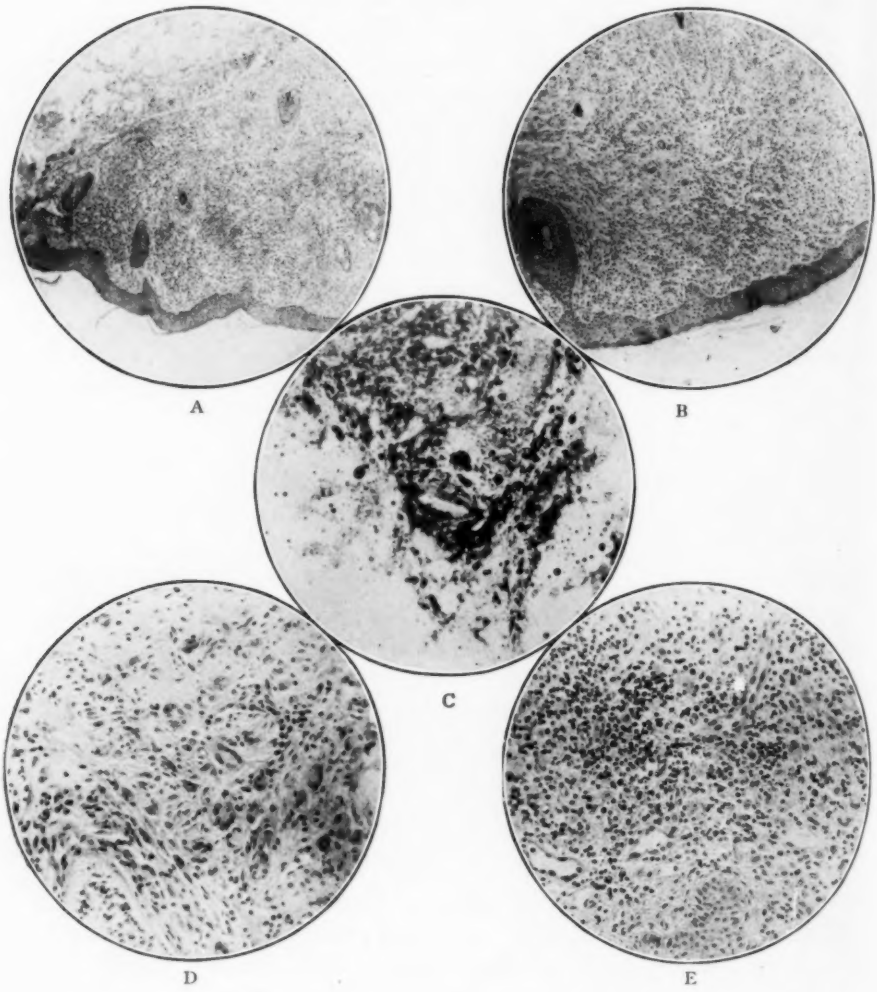


Fig. 8.—Photomicrographs from lesion behind ear consisting of early xanthomatous elevation and a portion of the chronic erythema; shows gradual transformation of latter into xanthomatous elevations: *A*, low power magnification of erythema showing marked cell infiltration, no multinucleated cells and no fibrosis as yet; *B*, low power magnification of region of xanthomatous fold showing many multinucleated xanthoma cells and beginning areas of fibrosis; *C*, frozen section of region of erythema showing lipoidal accumulation in and around dilated thickened blood vessel wall deep in the subpapillary layer; *D*, high power magnification of same areas as *B*; *E*, high power magnification of same areas as *A*. Note the dilated papillary and subpapillary blood vessels, absence of multinucleated cells, presence of mononucleated xanthoma cells and prefibrotic cell infiltration.

endothelial cells and infiltration of the contiguous cells, and suggested the action of some irritant circulating in the blood. This irritant was evidently the fatty substance which seemed to be eliminated from the blood, infiltrated the endothelial cells, the cells around the blood vessels and the tissue spaces themselves, causing the formation of the xanthomatous cells. The irritative process did not stop but led to fibromatosis and the gradual transformation of a soft cellular proliferation to a hard fibrous mass. In view of the fact that this patient at the present time has no hypercholesterinemia, it is not hard to conceive that the irritative process stopped in the stage when only erythema was present in one location, although it had proceeded to definite tumor formation in other locations, either as a result of trauma or increased capillary circulation.

DISCUSSION

Dr. J. Frank Schamberg, Philadelphia: The case just presented by Dr. Greenbaum I think is a most remarkable one. I had the opportunity of presenting this case two years ago before the Philadelphia Dermatological Society, since which time there has been progression of many of the lesions. The patient at that time presented the lesions of xanthoma tuberosum, but in addition there is now a pronounced erythema of the face which gives almost the color of the port wine stain, and associated with this is a parapsoriasis en plaque. It is doubtless an erythema perstans.

Dr. Greenbaum is probably correct that the erythema is a preceding stage of the xanthoma, that there has been a deposit of the cholesterol and then the combination with the connective tissue cells and the formation of the xanthoma cells. If one looks at the slides, he can see the granules stained with sudan III on the area. It is possible that there is some relationship in the pathogenicity between the eruption on the body and the xanthoma on the face, but I think no definite declaration can be made with respect to this. I think the question must remain open. So far as my experience goes, and the literature on the subject, the case stands out comparatively unique. The existence of the erythema on the skin in which cholesterol is first deposited and then xanthoma develops I believe is unique.

Dr. Fred Wise, New York: In regard to treatment, I wonder whether Dr. Greenbaum has considered a more drastic effort toward the cure of this eruption. Whitehouse, about a year ago, presented at the New York Dermatological Society a patient who had universal xanthomatosis. This patient went to Vienna, where a prominent dermatologist (I believe it was Riehl) administered large doses of iodipin and cured him. The histologic sections studied in Vienna showed a diffuse xanthomatosis. It is possible that iodipin might help this patient, or some other form of iodin medication.

Dr. Sigmund S. Greenbaum, Philadelphia: I simply wish to add that what I did here was to destroy the actual lesions around the eyelids, mouth and other parts, with the actual cautery, on the basis that since the man did not have a hypercholesteremia there could be no recurrence. I put him on a cholesterol-free diet and turpentine in addition. I intend to give him cholesterol by mouth, in conjunction with other experimental work on rabbits, to determine whether his cholesterol metabolism is normal, or as a cholesterol test, just as we perform the glucose tolerance test.

ICHTHYOSIS CONGENITA

WITH UNUSUAL COMPLICATIONS *

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From available literature, it would seem that generalized congenital hyperkeratoses have been recognized and recorded from early times. For many years, unquestionably, certain of the cases reported under the caption "ichthyosis congenita" included not only this condition as we now know it, but also cases which more recent study has identified as separate clinical entities. In view of these more exact classifications, the following case, presenting mixed characteristics and complications not ordinarily associated with ichthyosis congenita, and, finally, affording the opportunity of necropsy study, would seem to merit a somewhat detailed report.

REPORT OF CASE

History.—The mother of the patient, who presented herself at the Salvation Army White Shield Home, April 5, 1923, and was assigned to the service of Drs. Sheldon and Patrick, was a girl aged 18, obviously low in the intellectual scale. She could give no especially satisfactory history. She said that she was of American parentage, had lived her entire life in an isolated mountain district, and, so far as her knowledge went, knew of no familial disease or tendencies. Her father was living and apparently well; her mother had died "a long time ago." Up to the time of her entrance into the hospital she had never left her home for any length of time, being confined there by housekeeping duties for her brother and father. Her entire associations had been with these two, and she said that she was unable to tell which one was responsible for her pregnancy. This was the first pregnancy, and apparently had progressed uneventfully.

Examination of the Mother.—This revealed a poorly nourished, slightly anemic woman. Other than the striae, areolar pigmentation and the other usual changes coincident with pregnancy, the dermatologic examination was negative. Likewise, there were no noteworthy physical findings. The Wassermann test, urinalysis and blood examinations were negative. Briefly, the case was that of an uneventful pregnancy now at full term, with the unusual history noted in the foregoing. The patient was delivered the morning of April 8, and immediately the unusual appearance of the child attracted the attention of the attending physicians.

Examination of the Child.—This revealed a fully developed female infant of most unusual appearance (Fig. 1). The entire body gave the impression of

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* From the Department of Dermatology and Syphilology, University of Oregon Medical School.

* Read at the Forty-Eighth Annual Meeting of the American Dermatological Association, Washington, D. C., May 4, 5 and 6, 1925.

being encased in an extremely tight, perfectly fitting covering of heavy waxed or oiled paper. So marked was this over the hands, feet, fingers and portions of the legs, arms and face that the skin seemed at the point of bursting momentarily. Apparently the musculature of the parts was unable to overcome this resistance, for the joints were in semiflexion, and the child lay motionless except for feeble respiratory efforts. When motion occurred, and over the flexors of the hands and the feet, where occasional efforts at motion went on, fissures were making their appearance. More minute examination revealed a scalp sparsely covered with hair, but having the same peculiarly glazed or waxed over appearance. Neither eyebrows nor eyelashes could be seen with the naked eye, and an ectropian was developing. The nares were flattened as though by a tight

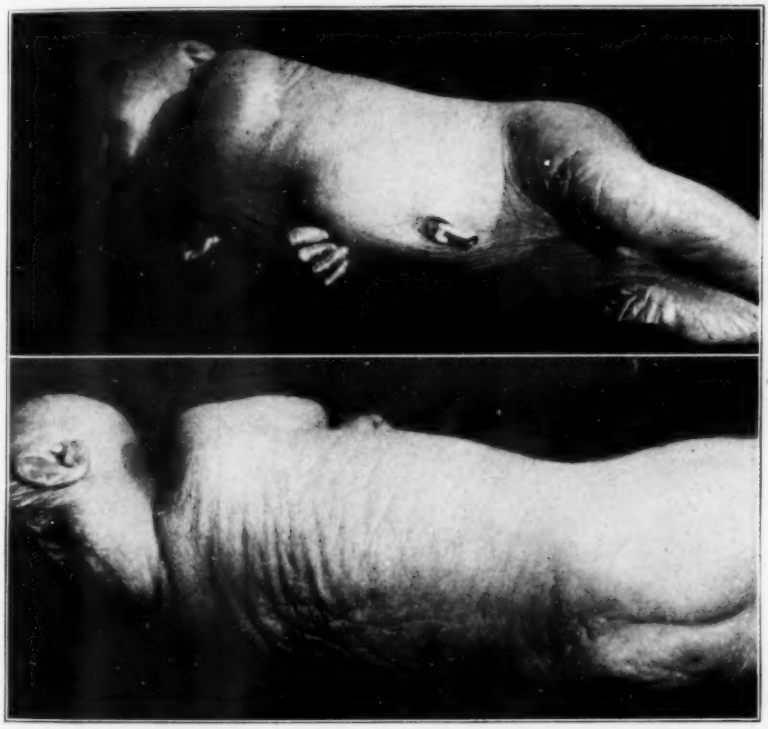

Fig. 1.—Patient second day after birth. Note glazed appearance of skin, semiflexed position of limbs and beginning fissures on trunk and limbs.

band, making nasal breathing almost impossible. The lips moved but slightly, and the child was unable to nurse (Fig. 2). The torso had the same waxed paper or shellacked appearance, but not to the same degree as the extremities. Here and there a fold of skin could almost be taken between the thumb and finger, but in spite of this diminished elasticity, the respiratory movements of the chest seemed most difficult, and resulted in the formation of fissures along the costal lines. As noted in the foregoing, the extremities were practically motionless, and the skin appeared as though stretched to the bursting point. Palpation of the extremities, especially of the fingers and toes (Fig. 3), gave

the hard, firm resistance of a process of sclerosis involving the skin and subcutaneous tissue with firm fixation to the underlying bony structures. There was no especial erythema or mottling; in fact, over the fixed joints was the whitish appearance of pressure anemia. In addition, no follicular openings could be made out. The child uttered an occasional feeble cry, accepted nourishment only occasionally by means of a dropper, and had, from birth, shown a subnormal temperature curve. Treatment was purely symptomatic, and consisted of frequent anointing with oil, and an attempt to introduce sufficient nourishment and sustain normal temperature.

Progress of the Case.—The subsequent course of the case was essentially that of steady progression in the process before described. By the end of the third week the picture had become even more pitiful. Marked ectropion had become established, the eyelids being practically immobile. In a similar way, the lips were fixed and open. The fissures on the body resulting from respiratory movements, superficial when first seen, had now become deeper, and were

Fig. 2.—Closer view of upper portion of the body, showing changes similar to those in Figure 1.

the starting points of large masses of desquamating epithelium. Similar desquamation had begun on the hands and feet, but to a less extent. In these locations, however, the apparent induration of the subcutaneous tissue, and its fixation to the underlying bony structures seemed more marked than before. At this time the patient's weight, which was 7 pounds, 2 ounces (3.2 kg.) at birth, had dropped to 5 pounds, 10 ounces (2.5 kg.). The patient was obviously weaker, and diarrhea had developed. The average temperature was still subnormal. By the fourth week the patient was becoming progressively weaker, the gastrointestinal condition was more severe, and there was a continued increase in the severity of the cutaneous condition. It was at this time that, in addition to the glazed, hyperflexed appearance of the fingers and toes, a peculiar, dark discoloration was beginning on the index finger and the thumb of the right hand. There was no secondary infection; the parts were cool, and the process seemed dependent on an underlying vascular pathology. Death occurred during

the sixth week, following the development of a respiratory infection of four days' duration. This was the only time during which the temperature was elevated, and it was accompanied by physical signs of pneumonia. The condition of the skin was changed only in degree. The scalp was sparsely covered with hair, ectropion was more marked, the tongue being the only part of the head remaining mobile (Fig. 4). Over the entire trunk, arms and legs, innumerable and deep fissures were present, giving rise to large desquamating plaques,

Fig. 3.—Appearance of feet two days after birth. Note glazed, waxed paper appearance and beginning fissures.

Fig. 4.—Patient just prior to death. Note fixation of eyelids, lips, fingers, etc.

up to the size of the patient's palm (Figs. 5, 6 and 7). In brief, the waxed paper appearance still persisted and gave the appearance of cracking in places, preparatory to exfoliation. The fingers and toes (Fig. 8) retained their talon-like flexion, and the boardlike feel and fixation. The changes involving the right index finger and thumb, before mentioned, had now become gangrenous (Fig. 9).

Necropsy.—Other than the cutaneous changes and terminal bilateral pneumonia, postmortem examination revealed no unusual gross pathology. Microscopically, the most striking features of the examination were found in the suprarenals, the thyroid and the skin. There were no significant changes in the other endocrine glands examined. Unfortunately, the thymus, pituitary and hypophysis were not included in the material taken. Examination of the viscera, brain, etc., was negative, or presented only those changes concomitant with a terminal infection of this type. The thyroid and suprarenals presented a striking picture of marked hypoplasia. In the thyroid (Figs. 10 and 11), not only did this change include a deficiency in colloid for a normal child of the patient's age, but the hypoplastic change seemed to extend throughout the gland. Peculiarly enough, this same hypoplasia was marked in the suprarenals (Figs. 12 and 13), but did not include the other internal glands examined. Sections of the skin were taken from the trunk, the extremities and the scalp. Unfortunately, because of conditions over which I had no control, the portion from the gangrenous area was not saved. With the exception of mild differences, the

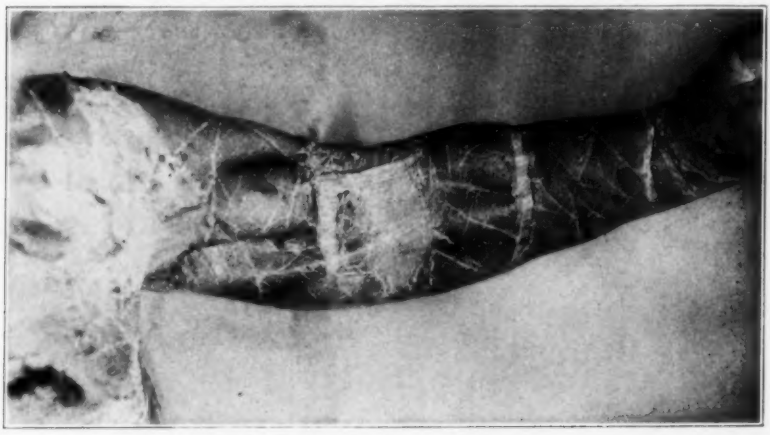

Fig. 5.—Note increased depth of fissures and extensive exfoliation.

sections of skin studied from various parts of the body presented similar changes. The most striking change was that of a marked thickening of the stratum corneum to several times normal thickness (Figs. 14 and 15). This apparently arose from a fairly definite granular layer. The horny layer took a fairly homogeneous deep stain, but in practically every section presented toward the surface the fragmentation, splitting and separation clinically evident on inspection. It had every appearance of a more or less incomplete, hyperplastic keratinization cycle. In some sections from the scalp (Fig. 16), this layer seemed actually to include portions of the hair, the hairs themselves in many instances showing fragmentation and a peculiar appearance of incomplete differentiation found in embryonic hairs. The stratum lucidum was not evident as such. The granular layer, contrary to the observations of other writers, was present in the sections examined. The cells did not have the usual appearance, and the layer was not well defined in places. It passed rather abruptly into the hyperplastic horny layer. The stratum malpighii was increased in thickness in but few sections. Even on close observation, this seemed more

apparent than real because of the lengthening of the interpapillary tufts. The cells in the layer were well formed and gave no especial clue as to the cause of the hypertrophic process involving the overlying horny layer. The stratum germinativum presented no striking changes except evidence of marked activity and of rapid division. The most striking change in the corium was to be seen in the marked widening and dilatation of many of the sweat coils. The sebaceous glands did not seem particularly affected. The fibrous elements of this layer presented no extreme changes, and, likewise, the lymph and blood vessels showed no particular variation except that of dilatation in certain sections.

COMMENT

It is not within the province of this study to detail the numerous writings appearing under the title "Ichthyosis Congenita." From such a perusal, however, and a study of the changes presented in the case in

Fig. 6.—Note fissuring, exfoliation, crinkled, waxed paper appearance.

question, a brief résumé of certain of the subdivisions of the congenital hypertrophies seems not to be out of place. Perhaps most extreme in degree is that group of cases now referred to for the most part as "harlequin fetus," "keratoma diffusum," "intra-uterine ichthyosis," etc. Such cases may be found in the writings of Lebert,[1] Caspary,[2] Sutton,[3]

1. Lebert: Ueber Keratose, Breslau, 1864.

2. Caspary, J.: Ueber Ichthyosis Foetalis, Vrtljschr. f. Dermat. u. Syph. **18**:3, 1886.

3. Sutton, J. B.: A Case of Generalized Seborrhea, or "Harlequin Foetus," Tr. Med.-Chir. Soc., London, 1886, p. 291.

Cabot,[4] Daniel and Cordes,[5] Sherwell,[6] Elliott [7] and others. They make up that group of cases in which at birth the entire body is covered by thick, horny plates of hypercornification; extreme degrees of ectropion and immobility are found; the patients lie motionless and practically helpless, and early death ensues from inanition, subnormal temperature, or inability to take properly or to assimilate nourishment.

The second type, which is still usually mentioned in this connection, but whose place is generally agreed to be elsewhere than in this category, is that group of cases first discussed by Hebra and Kaposi,[8] and later by Tommasoli.[9] These cases are listed under the term "ichthyosis sebacea" or "seborrhea squamosa neonatorum." Most modern writers,

Fig. 7.—Note fissuring and waxed paper appearance.

4. Cabot, J.: A Case of Ichthyosis Congenita with Some Unusual Features, M. Rec. **48**:10, 1895.

5. Daniel, A. S., and Cordes, L.: Case of Fatal Fetal Ichthyosis, J. A. M. A. **35**:1801 (Oct. 27) 1900.

6. Sherwell, S.: Ichthyosis Congenita (So-Called Harlequin Foetus). History of Case Still Living, J. Cutan. & Genito-Urin. Dis. **12**:385, 1894.

7. Elliott, G. T.: Two Cases of Intra-Uterine Ichthyosis, J. Cutan. & Genito-Urin. Dis. **9**:20, 1891.

8. Hebra and Kaposi, quoted by Stelwagon, H. W.: Ed. 8, Philadelphia, W. B. Saunders Company., 1916, p. 593.

9. Tommasoli, M.: Ein Fall von Ichtyosis Sebacea-Neonatorum. Vrtljschr. f. Dermat. u. Syph. **18**:456, 1886.

however, agree that these must be considered in reality but an abnormal accumulation of vernix caseosa continued a short time after birth. Such cases usually present an encrusted and frequently fissured integument, having a tightened, stretched appearance. The subsequent history of viability and an examination of the sebaceous mechanism, as brought out

Fig. 8.—Note glazed, rigid appearance of toes.

Fig. 9.—Gangrenous process involving the right thumb and forefinger.

by Elliott,[7] Caspary [2] and others, rather fail to substantiate the original contentions regarding the underlying pathology.

Finally, because of the clinical characteristics of the subject of this study, must be included that group of cases described especially by

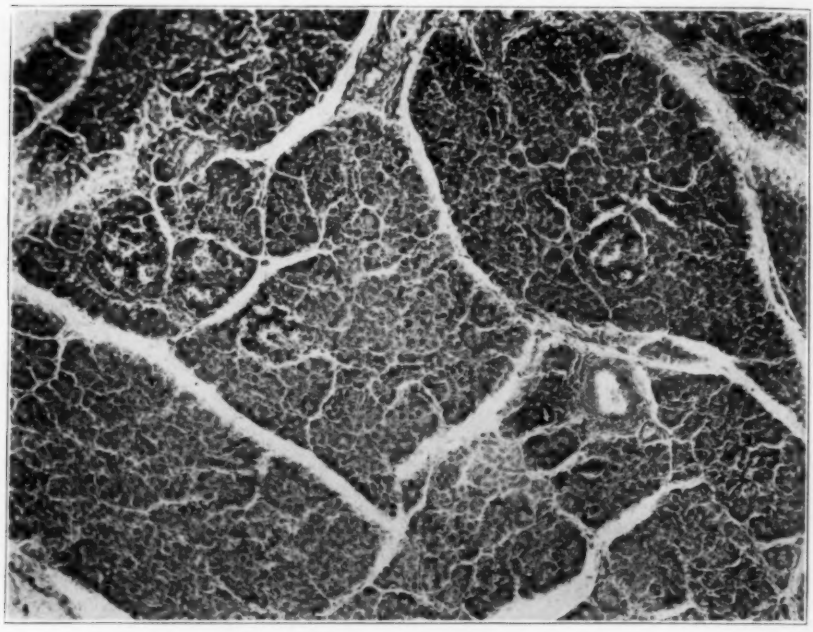

Fig. 10.—Hypoplastic thyroid, low power magnification.

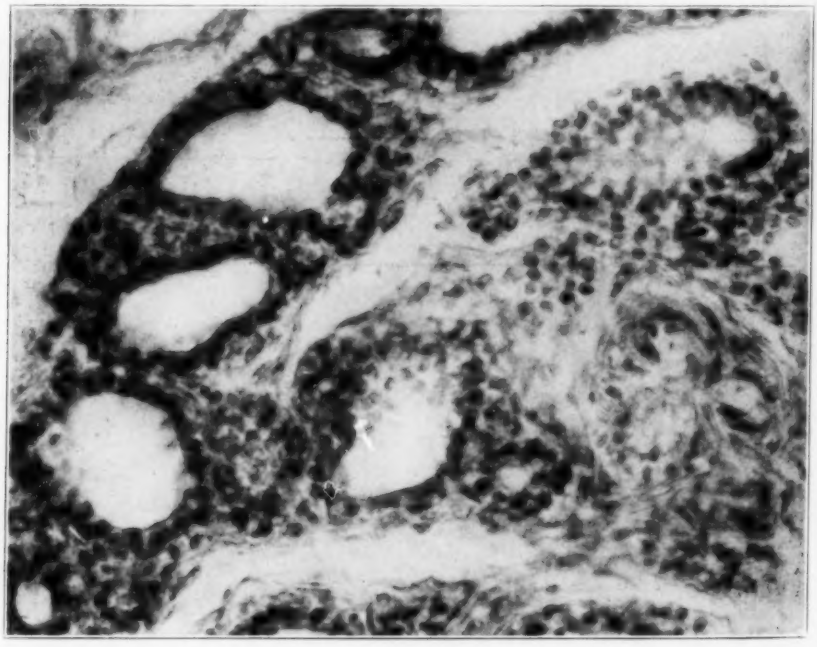

Fig. 11.—Portion of thyroid, high power magnification.

Hallopeau,[10] Gross and Török,[11] and Meneau [12] abroad, and mentioned by Bowen [13] and Ormsby [14] in this country. Bowen, in particular, has called attention to the possible abnormal persistence of a fetal membrane as a plausible explanation of the changes noted in certain reported cases. According to this author, in certain lower animals a "distinct outer membrane covering the hair development of the animal" is present. To this is given the name "epitrichium." Apparently this exists as a distinct membrane in certain mammals, but in others, including man, is represented by an upper layer of cells differing histologically from those below, this in turn being called the "epitrichial" layer. Examination of

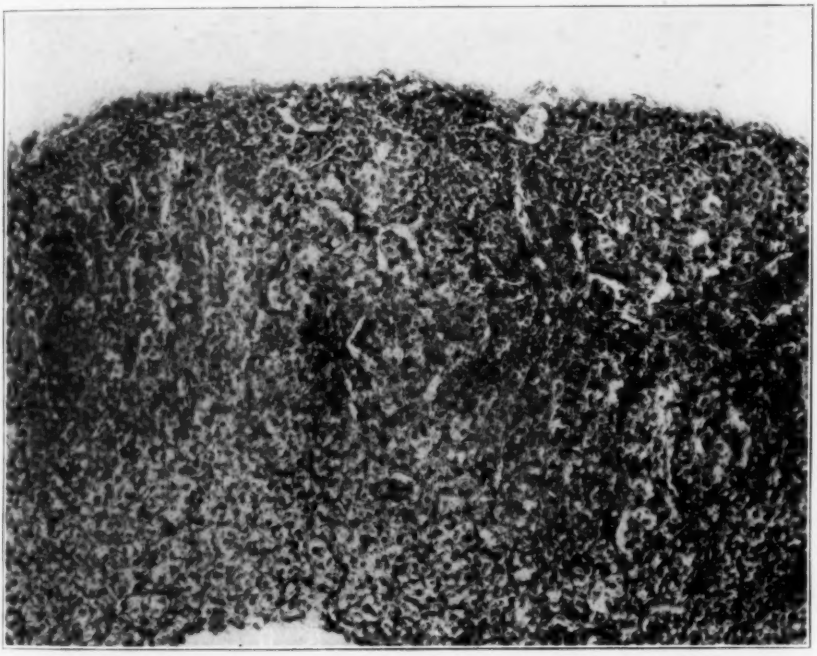

Fig. 12.—Portion from suprarenal, showing hypoplastic changes similar to those found in thyroid.

10. Hallopeau, M.: Sur une ichthyose avec hypotrophie simulant ume sclerodermie, Ann. de dermat. et syph. **3**:1016, 1895.

11. Gross, J., and Török, L.: Un cas d'exfoliation lamelleuse des noubeau-nes, Ann. de dermat. et syph. **3**:1041, 1895.

12. Meneau, J.: De l'ichthyose foetale dans ses rapports avec l'ichthyose vulgaire, Ann. de dermat. et syph. **4**:97, 1903.

13. Bowen, J. T.: The Epitrichial Layer of the Epidermis and Its Relation to Ichthyosis Congenita, J. Cutan. & Genito-Urin. Dis. **13**:485, 1895.

14. Ormsby, O. S.: Ed. 2, Philadelphia, Lea & Febiger, 1921, p. 490.

human embryos reveals this epitrichial layer (Fig. 17) made up of peculiarly shaped, rounded, puffed out cells, and sufficiently definite to be recognized as a separate histologic layer. The close resemblance of this layer in the human embryo to the cellular components of the epitrichium of certain animals seems strong presumptive evidence that they must be considered homologous histologic structures. Further, the partial horny tissue that covers the fetal nail, the eponychium, seems, according to Bowen, to be part of the epitrichial layer, as a direct connection between the eponychium and the peculiar cells of the epitrichial layer

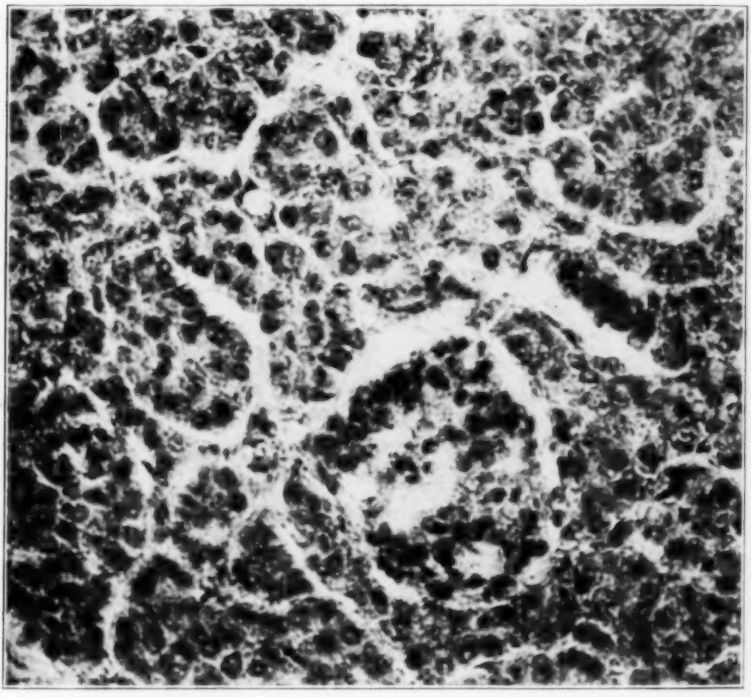

Fig. 13.—Portion of suprarenal, higher power magnification.

can be traced at certain times during prenatal life. The layer is usually entirely lost by the sixth fetal month, but in embryos of 5 months, after the nails are exposed by the loss of the epitrichial layer, a heaping up of horny cells is found at the nail border. Apparently this is produced by the persistence of the epitrichial layer in this location and by a keratosis of the cells making up this layer. Such a persistence of the layer, involving the entire integument and enduring after birth might, it would seem, as Bowen suggests, explain certain features of the case under

discussion. Such infants when born are invariably described as giving the impression of being encased in an oiled or waxed paper coating. Subsequently, during the early weeks of life, fissures appear, giving rise to marked exfoliation over the entire body surface. Ectropion, articular fixation and general immobility are not present to any marked degree. Following the exfoliative process, a normal integument is revealed, and the life of the child is not visibly affected by the primary cutaneous abnormality.

From the foregoing rather brief review of the diagnostic requirements on which is based the classification of cases in any one of the before-

Fig. 14.—Section of skin showing typical changes in horny layer.

mentioned three groups, the case under discussion must be considered unique in the characteristics presented. It would rather appear both to lack and to include certain essentials of the first and last reviewed. The thick, horny plates, the criss-crossing of deep and extensive fissures and the alligator appearance of definite ichthyosis congenita are lacking; on the other hand, the generalized, impeded mobility, the ectropion, the partial fixation of the lips, the subnormal temperature and the obvious incompatibility of the condition with life, make for its inclusion under such a head. At the same time, the exactly typical and, from other

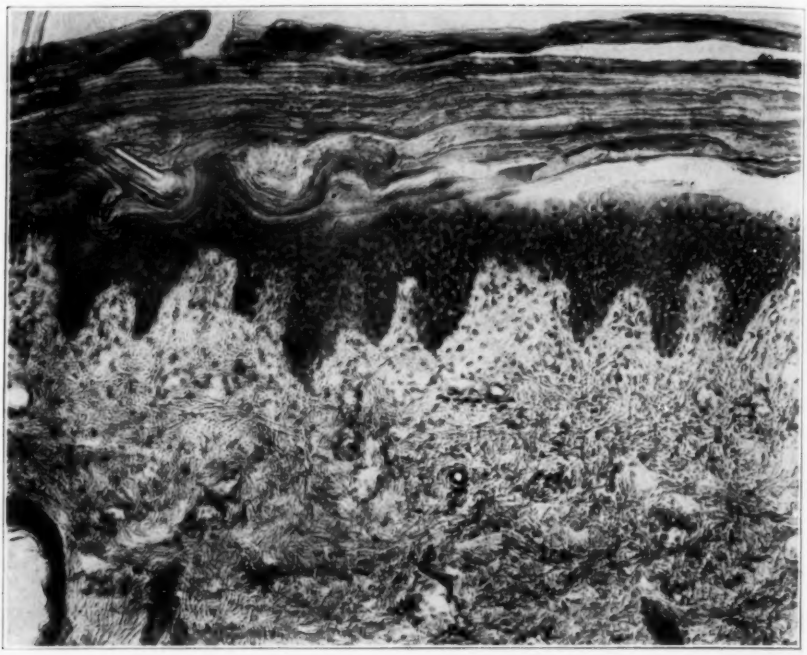

Fig. 15.—Section of skin from trunk, showing hypercornification, desquamation, etc.

Fig. 16.—Section from scalp showing fragmentation and tending toward inclusion of hairs.

descriptions, entirely characteristic oiled or waxed paper appearance, the development of moderately deep fissures and subsequent exfoliation, seem most easily explained by the epitrichial theory of Bowen.

In addition to this unusual picture was the gangrenous change in the hand. As mentioned, this was first noted at the fourth week, and it progressed up to the time of death. Whether this is to be explained by an impeded vascular supply secondary to what was clinically a sclerosing infiltration of underlying tissues, or whether the presence of forced contracture gave rise to the process, unfortunately cannot be decided microscopically. From available data it can only be said that such a complication has not been recorded previously in connection with studies of this disease.

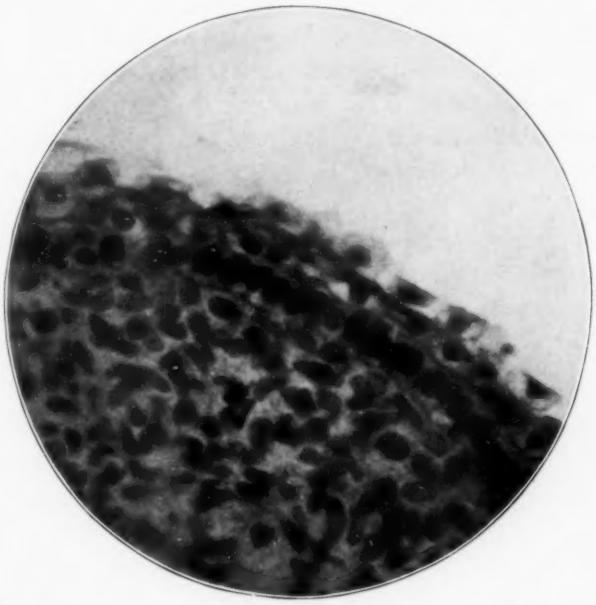

Fig. 17.—Section of skin from a 5½ month's fetus. Note outer layer of puffed-out, balloon-like cells described by Bowen.

Finally, to be mentioned in connection with the foregoing case, are the striking changes in the thyroid and suprarenal glands. The present trend of etiologic evidence, as expressed in most modern texts, attributes the most important rôle to heredity. To this theory, it must be conceded, such observations as those by Kaposi,[15] Crocker,[16] Frick[17] and others,

15. Kaposi, M.: Case Demonstration, Ann. de dermat. et syph. **3**:686, 1895.
16. Crocker: Case Demonstration, Brit. J. Dermat., 1895, p. 217.
17. Frick, W.: Three Cases of Ichthyosis in One Family, J. Cutan. Genito-Urin. Dis. **15**:19, 1897.

at present lend much support. Heredity, however, merely implies familial succession and is not an ultimate etiologic factor. However, the frequent mention of thyroid extract in the therapeutic management of such cases would seem to be indicative of a certain trend of opinion based on endocrine influences. Fox,[18] especially, expresses such a view regarding a case presented before the Royal Society, and quotes similar opinions by other contemporaries. Moore and Warfield [19] likewise found an almost complete lack of colloid material and alveolar formation in the thyroid gland at necropsy in a case of ichthyosis congenita. Finally, Kaposi expresses himself as of the opinion that the condition appears to be an anomaly of the nutrition of the skin, especially involving the epitrichium and fatty layers. The changes noted in the case under discussion lend favor to such a view. While the preponderance of evidence at present favors the possible influence of heredity, at the same time heredity cannot be considered a factor comparable to endocrine deficiency. Necropsy examinations are not so frequently recorded, yet when the time arrives that such statistics have been obtained, it would seem probable that the rôle of the thyroid in cutaneous metabolism, either directly or by virtue of its influence on the endocrine balance, may well assume a place of greater import. Suffice it at present to say that a complete etiologic explanation of these cases is not at hand, but rather rests on the results of future necropsy study, combined with the clinical facts that are now known.

CONCLUSION

1. In the foregoing is reported a case which includes clinical characteristics both of ichthyosis congenita and so-called "colloidal skin."

2. Such a clinical combination has not hitherto been recorded, and definite cases of colloidal skin are practically wanting in American literature.

3. The existence of the epitrichium in certain mammals and the presence of its analogy in human embryos would suggest the possibility of the persistence of this membrane as a partial explanation of the foregoing picture.

4. The rôle played by the endocrine glands or their interrelation in the production of such cutaneous anomalies is suggested, and the importance of further study directed toward such an end is emphasized.

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18. Fox, C.: Case Demonstration, Brit. J. Dermat. **23**:112, 1911.

19. Moore and Warfield: Relation of Thyroid to Ichthyosis, Abstr. J. A. M. A. **46**:1130 (April 14) 1906.

DISCUSSION

DR. OLIVER S. ORMSBY, Chicago: I have had an opportunity to study these cases somewhat and am very much pleased to have Dr. Kingery's explanation as to the cause of them. I was always interested in Bowen's explanation and presumed it was a retention of the epitrichial layer. I think there is no doubt that this is a form of ichthyosis. In one case which I studied for some time the "collodion skin" disappeared within a few weeks. In another case the patient died very soon, and the case could not be studied. It is difficult to prove whether the explanation of Bowen is correct, but it is an interesting explanation and even if it is not correct, it may be that the thyroid is concerned in its etiology.

PSORIASIS IN A FULL-BLOODED NEGRO*

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NEW YORK

Psoriasis, according to the statistics of the American Dermatological Association, constitutes about 2.7 per cent. of all skin diseases. It is fairly common in the white race, so much so as to attract but little attention. Among colored people, on the contrary, this dermatosis is so rare that some American dermatologists have even disputed its existence in the negro.

REPORT OF A CASE

R. B., aged 42, an American negro (full-blooded), was admitted to the syphilis clinic of Bellevue Hospital, Feb. 11, 1924. He gave a history of chancre twenty-three years previously, the scar of which was still present on the glans penis. Whether he had had secondaries or any treatment for same was not noted. It may be stated that he had no external evidence of syphilis.

Examination revealed a generalized squamous eruption, most marked on the extensor aspects of the body and involving the scalp and face. This eruption was intensely pruritic. The lesions were perfectly dry and the scales were "silvery white," as described in most textbooks. The location, distribution and appearance of the lesions were typical of psoriasis. The patient stated positively that he had never before had a similar eruption.

The Wassermann test, on February 14, gave a $++++$ reaction. Before treatment was instituted, the patient was presented at the Manhattan Dermatological Society, February, 1924, with a diagnosis of psoriasis. This diagnosis was not accepted by the majority, but the diagnosis of secondary syphilis was favored in spite of the history of chancre twenty-three years previously, scar of chancre, perfect symmetry and intense itching.

To exclude syphilis I gave the patient 6 intravenous injections of arsphenamin and 6 intramuscular injections of mercuric salicylate, which did not produce material improvement in the skin lesions.

One month later the patient was again presented at the Manhattan Dermatological Society, and the majority then concurred with the diagnosis of psoriasis.

Dr. D. L. Satenstein suggested and made a biopsy, and reported that it showed a true case of psoriasis.

The patient was also presented at the dermatological section of the New York Academy of Medicine, May 6, 1924, when the diagnosis of psoriasis was again agreed to.

As the question of diagnosis was settled, the patient was given local treatment with equal parts of ammoniated mercury and tar ointment, as a result of which he showed marked improvement. He discontinued his visits.

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* From the Department of Syphilology, Bellevue Hospital.

Fig. 1.—Anterior and posterior view, showing numerous white, scaly typical psoriasis lesions.

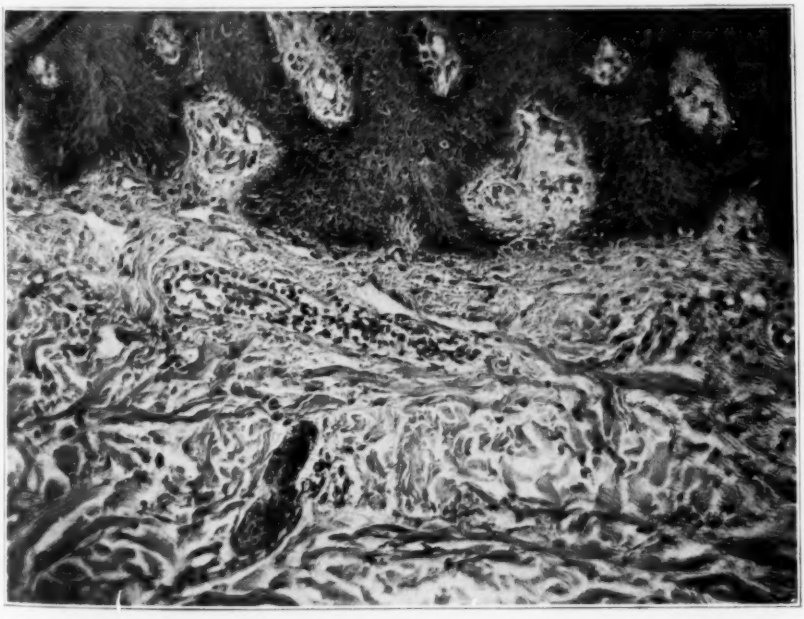

Fig. 2.—Histology.

On October 14, the patient returned with an exacerbation of the skin lesions much more pronounced than the first, and complained of an intolerable itching. He was again presented before the Manhattan Dermatological Society at the October meeting, with a unanimous acceptance of the diagnosis of psoriasis.

Corium: The papillary and subpapillary layers showed infiltration with a cellular exudate in the lymph spaces and following the course of the blood

Fig. 3.—Patient showing recurrence in October, 1924.

vessels. The exudate was composed chiefly of small, round mononuclear cells of the lymphocytic type.

Epiderm: The prickle cell layer showed hyperplasia and slight edema. Defective keratinization was shown by the complete absence of a granular layer and collections of nucleated cells in the corneum.

The histopathologic picture was consistent with the diagnosis of psoriasis.

REVIEW OF THE LITERATURE

It is significant that Rufz,[1] 1859, had never seen psoriasis at Martinique.

Morison [2] gave figures of twenty-six cases of psoriasis in 500 white persons against two cases in black males and four in female mulattoes in a total of 500 negro patients.

Dr. Corlett [3] of Cleveland stated that he had never seen psoriasis in the negro, and at the same meeting Dr. George H. Fox [4] of New York said that he had never been able to obtain a photograph of well-marked psoriasis in a negro. He added that possibly the naked savages of Africa were free from psoriasis on account of the exposure of their skin to sunlight, and that the negroes of North America may have inherited this peculiarity.

Dr. Howard Fox,[5] in 1907, in an article based on statistics collected in the southern states, gives forty-nine cases (0.0222 per cent.) of psoriasis occurring in 2,200 white persons against ten cases (0.0045 per cent.) in an equal number of negroes. He does not state, however, whether the patients were full-blooded negroes or of mixed blood.

In 2,000 consecutive cases of skin diseases in whites and negroes, Hazen [6] found eighty-two cases of psoriasis in whites as against eight in negroes. He was informed by Chalmers that the latter had seen a number of instances of psoriasis in native Africans.

At the Pittsburgh Dermatological Society meeting in November, 1923, Dr. Wright [7] presented for Dr. J. F. Schamberg a case of psoriasis in a negro. "This case was presented because of the rarity of the combination of psoriasis and a colored person." But in his article on psoriasis Schamberg [8] states that he has never seen a case of psoriasis in a full-blooded negro, which would indicate that the case presented by Dr. Wright was probably that of a mulatto. At the Pittsburgh meeting, Dr. Knowles stated that he had seen "a few cases" in the University Hospital, but that they usually occurred in the lighter colored negroes.

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1. Rufz: Note sur la frequence et la diversite des maladies de la peau a la Martinique, Bull. de l'Acad. imper. de méd. **24**:1051-1072 (June 28) 1859.

2. Morison, R. B.: Personal Observations on Skin Diseases in the Negro, M. News **53**:439-444 (Oct. 20) 1888.

3. Corlett (in Hyde): Brit. M. J. **2**:839 (Oct. 6) 1906.

4. Fox, G. H. (in Hyde): Brit M. J. **2**:839 (Oct. 6) 1906.

5. Fox, Howard: Observations on Skin Diseases in the Negro, J. Cutan. Dis. **26**:67-79; 109-121 (Feb.) 1908.

6. Hazen, H. H.: Diseases of the Skin, Ed. 2, 1922, p. 441.

7. Wright: Psoriasis in a Negro, Arch. Dermat. & Syph. **9**:398 (March) 1924.

8. Schamberg, J. F.: The Known and Unknown About Psoriasis, J. A. M. A. **83**:1211-1213 (Oct. 18) 1924.

In a discussion of Dr. Schamberg's paper, Fox [8] commented on this case that I have just reported, and stated that he had never seen an extensive eruption of psoriasis in the full-blooded negro until the past winter, when he saw this case and another of his own which showed a negative Wassermann reaction.

Dr. E. G. Lane [8] of Oklahoma City states that the American Indian appears to have the same immunity to psoriasis as the negro, and that in his observations of Indians he has yet to find the first case of psoriasis or anything simulating it, even the half breeds seeming immune.

Morison,[2] in 1888, writes that in psoriasis in negroes:

> The spots are covered with a more yellowish, less shining crust than in the white. When punctate, the crust is thicker than usual and is easily rubbed off. It then leaves a deep red, bleeding surface, the blood oozing freely from the minute vessels. As in other diseases of this race in which there is a sufficiently thick amount of epidermis removed, a white spot remains for a while after healing. In the orbicular form, many of the larger spots could still be seen after many years. It is not a common disease of the blacks and is easily cured. I have seen no relapses, nor had there been previous attacks as far as could be found out.

COMMENT

My observations differ from Morison's in three respects: 1. The scales were perfectly white, as in psoriasis in white people. 2. The patient had a relapse within two months. 3. The disease was not easily cured.

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THE SALICIN TREATMENT OF PSORIASIS

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In 1895, Radcliffe-Crocker[1] published a paper on the advantages of using sodium salicylate in psoriasis and some other diseases of the skin. Later he substituted salicin for the salicylate, as the latter is likely to upset patients in various ways, whereas salicin rarely, if ever, disagrees with them.

As I worked with the late Dr. Radcliffe-Crocker for about seventeen years, both in his hospital clinic and in his private practice, I shall discuss the salicin treatment of psoriasis and give details of his experience and of my own in the handling of the drug in order to obtain the best results. I would emphasize that salicin is not to be given in *any* manner, at *any* time and in *all* cases of psoriasis.

Salicin (salicinum) is a glucosid obtained from the bark of various kinds of willow (*Salix fragilis, S. purpurea, S. alba,* etc.). It was first introduced as a succedaneum for quinin (Leroux, 1830, Piria, Braconnot and others).[2] Salicin is available pharmaceutically as colorless shining tabular crystals, moderately bitter. It is not very soluble in water (1:28), which is a drawback. This must be borne in mind when prescribing it as a mixture. Roughly, one may reckon solubility at from 1 gm. to 1.2 gm. in 30 c.c. of water, so that pulverized tragacanth or any other suspending body must be added when dispensing larger doses. In my practice, I start with 1 gm. in 30 c.c. of water flavored with peppermint, increasing to 1.2 to 1.5 or even 2 gm. in 30 c.c. for each dose to be taken in plenty of water three times a day immediately after meals. There is something in the old medical tag "corpora non agunt nisi soluta," and in the case of salicin this is especially important as far as favorable results are concerned in psoriasis. Salicin is of no value if taken in the form of tablets. It is possible that physicians have been disappointed by poor results or no results if they have prescribed salicin in the solid form. I need hardly say that the salicin must be fresh, and I always indicate this when prescribing the drug.

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1. Radcliffe-Crocker, H.: Lancet, 1895, p. 1421; Brit. J. Dermat. **7**:229, 1895.

2. Robin: Dictionnaire de médecine, 1886. Kobert: Lehrbuch der Toxicologie **2**:144, 1906, with bibliography from 1860 to 1906.

In contradistinction to sodium salicylate, salicin is a safe drug and does not require the supervision that the former does, a great advantage when treating so obstinate a condition as psoriasis. Kobert[3] points out that very large doses (10 gm. and 30 gm. a day, that is, 150 grains and 450 grains) produced no ill effects in man except in some persons who exhibited a variety of symptoms. I merely mention this to show that the doses I have mentioned as indicated in the treatment of psoriasis are moderate and even small compared with the figures given by Kobert writing as a toxicologist.

The general condition of the patient with psoriasis must be considered. It is wise to examine the urine in every case, a procedure too often neglected, before prescribing an alleged specific like arsenic. Arsenic has a limited application in dermatology. In acute or subacute generalized psoriasis, arsenic is likely to do harm and should not be ordered. In such cases salicin has proved useful.

I could give the details of a number of cases, in which the patients have been under my care, but I shall give only those in one case which is instructive.[4] On March 28, 1912, a physician brought his wife to me for treatment of generalized psoriasis of from ten to twelve years' duration, which had been worse for the last six years. This was the spring offensive of the psoriasis, which was mainly punctate and nummular in type. The patient had been taking arsenic for four months at the time I first saw her. I prescribed salicin, 1 gm. in 30 c.c. of water, to be taken regularly and perseveringly. This is an important point, for if the salicin mixture is taken in a half-hearted, irregular manner, one cannot expect much in the way of results. In addition, I ordered occasional bicarbonate of soda baths at a temperature of 100 F., and as a local application, glycerin of lead lotion (1 part in 8 parts of rose water [30 c.c. to 230 c.c.]) When salicin is being administered, soothing local applications only are to be used. Therefore chrysarobin, tar and such preparations are not to be employed. This rule must be observed, as Radcliffe-Crocker and I found out by experience.

I had impressed on the physician that the salicin was to be given in the form of a mixture, but notwithstanding this he had given his wife tablets instead, and he informed me some time later that the salicin had caused diarrhea and had done no good. I told him to give the salicin in mixture form as recommended, and when this was done the psoriasis at once began to disappear, and in May, 1912, he reported: "You will be pleased to hear that my wife's psoriasis is slowly improving with the salicin in emulsion, which does not

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3. Kobert: Dictionnaire de médecine, p. 145. The late Dr. T. J. MacLagan of London used to give and advocate very large doses of salicin in acute rheumatism, as far back as 1874 (Lancet, 1876) with benefit; Fagge and Pye-Smith: Text-Book of the Principles and Practice of Medicine **2**:704, 1891. This has interesting tables. This book is a valuable and erudite work of reference, though now out of date, but none the worse for that. We can at times learn a great deal from our forerunners in medicine, who were great and patient clinical observers. As Osler put it: Medicina tota in observationibus.

4. Pernet: Private Case Book J., folio 99.

produce the ill effects of the tablets." In passing I may say that salicin in mixture appears occasionally to lead to a little constipation, but this can easily be remedied by a saline such as magnesium sulphate added to the mixture or taken independently early in the morning.

I saw the patient again at the end of April, 1913. She had been taking the salicin for a week for a fresh attack of psoriasis in the shape of numerous punctate and guttate lesions about the abdomen chiefly, and on the chest. Apparently, the eruption had followed an attack of tonsillitis and quinsy. The psoriasis was checked under treatment with the salicin mixture and cleared up.

In September, 1914, I saw her again. She had not had psoriasis for about six months. When I saw her in June, 1915, she told me that following a severe attack of influenza in December, 1914, she had had an extensive attack of psoriasis, which rapidly gave way to treatment with salicin, and she had been free from this condition for two months.

I did not see her again until October, 1917, when she came to me for loss of hair as a result of shock following a big explosion in a munitions factory in her neighborhood. She did not have psoriasis. In August, 1920, she came to me with another patient. She then told me that she had had not had psoriasis since the attack in 1914. In March, 1925, this patient brought her daughter to me, and she assured me that there had been no return of the psoriasis.

As the last attack of psoriasis occurred in 1914, this gives an interval of freedom of eleven years, the longest I have ever known in my practice. I can not say that she will never have another attack, nor that salicin will cure psoriasis; but salicin often checks and clears up acute and subacute attacks of psoriasis.

Salicin does not terminate eruptive attacks at once, but after a short time fresh elements cease to appear, and the earlier ones involute. Patience and perseverance are necessary.

OTHER TYPES OF PSORIASIS RELIEVED BY SALICIN

I have also found salicin useful in attacks of spreading psoriasis in children, adapting the dose to the age of the patient, and in young women. The elements of psoriasis in young women are generally paler in tint than usual and the scaling moderate, owing no doubt to the succulence of the youthful skin.

In suckling mothers, psoriasis may become acute and generalized as a result of lactation, so much so that the infant may have to be weaned. In such cases also salicin has proved most useful.

Another well-known type of psoriasis is that observed in adults, especially in the elderly. The rash is usually dull red, acutely inflammatory, scaly and widespread, rapidly involving large areas of the skin by coalescence. In these severe and extensive cases, the first indication is to put the patient to bed and to prescribe soothing, oily calamin applications, in addition to salicin internally in increasing doses, well

diluted and given regularly. Sometimes it may be necessary to add a little nux vomica to the mixture to be given three times a day in water after meals, thus:

℞	Salicin	1.2 to 1.5 gm.
	Liquid extract of nux vomica.....	0.06 c.c.
	Tragacanth powder	q. s.
	Peppermint water, Water } Equal parts.....	30 c.c.

Another acute generalized form of psoriasis is that associated with rheumatoid arthritis, usually observed in women. In these patients nutrition is lowered. Here also, I have found salicin helpful; not only is the rash moderated, but the joints also appear to be influenced beneficially in these obstinate cases. In old chronic patchy psoriasis, salicin is not effective.

ETIOLOGY

Although the essential etiology of psoriasis is unknown, I have long believed that it is probably of microbial origin. In favor of this view is its periodicity, with offensives in the spring and fall. The empirical results obtained with salicin are also in favor of this view, if, as it has been stated, salicin breaks up in the blood into salicylic acid and phenol.[5] The microbe may perhaps be of the filter passing, ultramicroscopic kind.[6] Civatte's recent researches into the histology of psoriasis point in the same direction, namely: that the lesion of psoriasis is more easily explained by the action of a virus or of a toxin coming from within.[7] The results of examination of the cerebrospinal fluid in psoriasis are also in favor of a constitutional microbial origin.

COMMENT

Finally, salicin is not prophylactic, so that it is useless to continue with the drug indefinitely after its effects have been obtained in an acute or subacute attack.

I have limited my statements concerning the use of salicin to certain definite clinical types of psoriasis in the hope that some of my readers may be induced to give it a fair trial in the conditions I have indicated.

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5. Radcliffe-Crocker: Diseases of the Skin, Ed. 9, London, H. K. Lewis, 1903, p. 338.

6. Pernet: Psoriasis, The Practitioner, September, 1914.

7. Civatte: Psoriasis and Seborrhoic Eczema: Pathological Anatomy and Diagnostic Histology of the Two Dermatoses, Brit. J. Dermat. **36**:476, 1924.

SO-CALLED HERXHEIMER REACTIONS*

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AND

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The Herxheimer reaction, first described in 1902, manifests itself in exacerbating syphilitic lesions associated with inflammatory changes which are most frequently produced by antisyphilitic therapy with mercury, arsphenamin or bismuth.

As the syphilitic lesions of the skin and mucous membrane are most pronounced, it is evident that the Herxheimer reaction is best known in association with these lesions.

The primary lesions, the localized secondary manifestations of syphilis, the severe skin affections in malignant syphilis and the tertiary lesions in general do not give such severe nor such readily recognizable reactions as the widespread exanthems of the secondary stage. In this stage, the reaction manifests itself in its most characteristic form, presenting lesions of the skin associated with hyperemia and edema. In a typical mild case, exanthems may appear immediately after the first treatment and "cure" when a sufficient dose has been administered. The most characteristic picture of the reaction includes an increase in the number of macules, the more pronounced elevation of the cutaneous lesions combined with the inflammatory reddening of the skin and the appearance of the bright red areola (especially when pustules are present) and urticaria-like lesions.

It may be stated that the blurring of a previously clear-cut syphilitic eruption after antisyphilitic treatment is the most usual manifestation of the reaction. The reaction as a rule disappears quickly, and in general it may be stated that the reaction subsides within a shorter time if the skin eruption appears promptly. The intensity of the reaction depends to a certain extent on the specific action of the antisyphilitic drug.

Arsphenamin treatment is likely to cause a more severe reaction, corresponding to the bactericidal action of the product. Subsequent to arsphenamin therapy, the reaction may appear within two hours and may subside after from six to eight hours. In cases of mercury and bismuth treatment, the reaction usually takes place after a longer period and persists for a greater time.

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*From the Frankfort University.

*Translated and read by title at the Section on Dermatology and Syphilis of the New York Academy of Medicine, February, 1925, by Dr. Mihran B. Parounagian, New York.

In cases in which the exanthems are obscure clinically the skin lesions may become acute after the administration of antisyphilitic medication, and the reaction therefore may have a diagnostic significance. For specific provocative diagnosis of skin lesions the most suitable method is an intramuscular injection of 1 c.c. of 10 per cent oily suspension of insoluble mercury preparation (mercuric salicylate) common bismuth salts or, best, silver arsphenamin, injected intravenously in doses of 0.15 gm. to 0.2 gm.

It is our experience that after the administration of mercury or bismuth in the doses mentioned, the reaction often appears within twenty-four hours, while in the case of arsphenamin it appears within twelve hours.

The reaction of the lesions of the skin and mucous membrane has a limited interest as compared with that of the viscera. As a result of several years' experience in the use of arsphenamin, it was ascertained that after the administration of arsphenamin, mercury and bismuth in sufficient doses, similar local reactions associated with hyperemia and edema as described above may appear in every organ of the body. Under certain conditions, antisyphilitic drugs may produce sudden changes which may endanger life. The consequence of the reaction, besides the intensity, depends also on the location of the lesions, namely, on whether or not a vitally important organ is involved.

The great variation in the symptoms is easily understood when the great difference in the structure of the involved organs is considered. The temporary swelling of the lymph nodes, the sensitiveness of the muscles, the swelling of the joints, accompanied by pain, the periosteal reaction (especially common to the tibiae), the periosteal hyperemia of the flat bones of the skull which directly or indirectly may involve the nerves running through the bones, the symptoms referring to the central nervous system and to the meninges, in cases of syphilitic involvement of these organs, the reaction of the heart and vascular system, the various signs and symptoms referable to liver and spleen (icterus and enlargement), the acute albuminuria—all are signs of a hyperemic, edematous, local reaction of the organ involved.

The moderate rise of temperature which is likely to occur after the administration of one or two injections corresponds to the same local reaction, except that in this instance the entire system is involved. They both belong to the same group of the so-called provocative Wassermann reaction (Milian-Gennerich).

The explanation of the reaction described is similar to that of the tuberculin, luetin or trichophyton reactions in which there is an intermediate action between the specific organism and the hypersensitive cells of the patient. In the sensitized tissue, through allergic and immune

processes a potential hypersensitiveness develops against these organisms. This potential hypersensitiveness is activated after the administration of specific drugs through their effect on the trypanosomes (stimulation of the living parasites, or liberation of large amounts of endotoxins). The final manifestation of this hypersensitive reaction is the development of hyperemia and edema in the tissues affected by syphilis. Besides the specific action, the effect of antisyphilitic therapy on the protoplasm may also play some rôle in the production of the reaction. This is especially true in the case of arsphenamin.

The nonspecific reactions which occasionally occur in nonsyphilitic skin diseases (such as psoriasis, lichen ruber planus, lupus vulgaris, lupus erythematodes, lepra, etc.) may be explained by this theory.

Though in rare instances the reaction mentioned may not be antisyphilitic, in the great majority of cases it is; and it is a manifestation of the effect of a certain group of chemicals on the hypersensitive tissues involved by syphilitic infection as a specific pharmacodynamic action.

News and Comment

NOTICE TO CONTRIBUTORS TO "DERMATOLOGY"

Attention is called to the following suggestions to writers, particularly to reporters of society transactions:

Do not use "lues" or "luetic" for "syphilis" or "syphilitic"; it is not good usage.

Do not use "salvarsan" or "606"; we use "arsphenamine."

Do not use *"Treponema pallidum"* or *"Spironema pallidum"*; we use *"Spirochacta pallida."*

Do not furnish reports of cases in society transactions which have been presented previously before another society whose transactions are published in the ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY.

Write complete sentences—not telegrams.

W. A. P.

Correspondence

ANTIQUITY OF SYPHILIS IN EUROPE

To the Editor:—I hesitate to add to the interminable discussion of the antiquity of syphilis in Europe, but since reading the interesting paper of Dr. Capper I am tempted to bring to your attention a case highly suggestive of a syphilitic infection that was described by the victim, a resident of Paris, in 1461.

It is true that the victim was a poet, and a notorious blackguard, jail bird, and even, during a period of financial embarrassment, major-domo of a low-grade bawdy house. And yet these irregularities that might, perhaps, discredit a serious syphilographer, seem to me to be the best of qualifications for a syphilitic autobiographer.

The literary patient in question is our old friend, Maitre Francois Villon, born in 1431, who disappeared in 1461. His more than kindly fictional biographer, Justin McCarthy, has represented him to us in "If I Were King" as a moral, even genteel, figure; but, truth to tell, he was an unmitigated rascal and an inveterate sensualist. His usual address was the city jail, relieved by general delivery on such occasions as the accession of a new king, etc. If there was syphilis to be had in Paris at the time, I think we may, without disrespect, assume that Master Francois acquired it, by fair means or foul.

His medical confessions are to be found in his last and greatest poem, "The Greater Testament," written the year he disappeared from this world's stage. He had recently been ejected from the prison of Melun by the grace of Louis XI, and had crept back to Paris, a sick and broken man, to the sanctuary of "la grosse Margot," where he found ignoble shelter while composing his masterpiece.

His allusions to his symptoms are to be found in stanzas 23, 60, 62, 165 and 173, and in the concluding ballad of the poem. I will not burden you with the labor of translating a quantity of medieval French, but will give you the sense of his allusions to his malady.

He describes himself as a "broken man, with skin darker than a mulberry." "I carry the voice and tone of an old man although in years I am but a young cockerel." "From my throat," he says, "I spit up gobbets of white phlegm as large as a tennis ball." He ascribes his malady quite bluntly to the opposite sex: "Love now and wenches I forswear, for death through them treads hard upon my heels." "I die," he says, with a smug groan, "a martyr to love." He refers feelingly to the fact that his hair, beard and eyebrows had all fallen out, leaving his head like "a peeled turnip." His genitalia (he is much more explicit in the original) were diseased, and his whole body was racked with pains.

It would seem a fair inference that a man of 30 years, of astoundingly vicious habits, who complained of a localized genital lesion, laryngitis, generalized alopecia, marked cutaneous pigmentation and severe bodily aches and pains, and who himself ascribed his condition to venereal causes, was in the throes of a fairly acute syphilis.

It is supposed that he died soon after writing the "Testament," as he dropped completely out of sight immediately thereafter.

His passing has been one of the great unsolved mysteries of literature; I would suggest that it was due to an authentic case of pre-Columbian European syphilis.

Yours very truly,

F. M. Campbell, M.D., Walla Walla, Wash.

Editorial Note.—Of course, this picture is not conclusive evidence of syphilis; the patient might, for example, have had laryngeal tuberculosis, which would have been more likely than syphilis to account for his sudden disappearance soon after this time. The cutaneous lesions might well be those of vagabondism; indeed the whole picture is one of "vagabondismus." Nevertheless, this dermatologic observation of one of the most interesting characters in literature is well worth recording.

Abstracts from Current Literature

Immunity in Cancer. F. C. Wood, J. A. M. A. **85**:1039 (Oct. 3) 1925.

In a thorough review of the literature of the subject, Wood finds that there is no evidence of immunity development in cancer. The evidence of animal experimentation pointing to the existence of cancer immunity must be rejected when subjected to critical study. Experiments in the radiation of transplantable tumors in animals likewise fails to convince. In this connection, Wood rejects Murphy's conclusions of lymphacytic activity against cancer. The rare cases of spontaneous regression of human tumors must be explained on some other basis than that of immunity.

The effect of radiation on cancer is a direct one on the cancer cell, and the secondary effects on the surrounding tissue have nothing to do with a curative result except in a purely mechanical way. The scar tissue that follows radiation of malignant tumors entraps those cells which escape a lethal dose, and prevents their dissemination. This has been proved by histologic study. The mechanical effect of the scar tissue, together with the lowered blood supply in all scars explain those cases of late recurrences in situ. There is no cogent evidence for, and much against, the conclusion that any process of immunization aids in the therapeutic efficacy of radiation.

Absolute permanence of cure by radiation therapy implies that the destruction of the tumor cells must be as complete as must be their removal by effective surgery.

Epithelioma and the Chronic Varicose Ulcer. L. C. Knox, J. A. M. A. **85**:1046 (Oct. 3) 1925.

Two cases of squamous cell epithelioma arising in crural varicose ulcers are reported, together with a collection of the recorded cases, which number only fifty-nine.

Epitheliomas arising in this manner run a slow course and metastasize late, but are in no sense benign. It is probable that chronic dermatitis rather than varicosities and altered blood supply initiate the malignancy. .

Michael, Houston, Texas.

Epithelioma of the Lacrimal Sac. R. A. Fenton, J. A. M. A. **85**:1128 (Oct. 10) 1925.

Fenton reports a case of secondary involvement of the lacrimal sac, the malignancy originating near the inner canthus several years previously. It had been unsuccessfully treated with roentgen-ray and radium. Excision and canthoplasty were performed, but was followed by a recurrence, which, however, has been stationary since a massive dose of radium was given.

Primary malignancies of the lacrimal sac are rare; the present case, one of secondary involvement, emphasizes the importance of thorough radiation of growths originating near the eye.

SEMINAL VESICLES FROM SYPHILITIC PATIENTS; HISTOLOGIC STUDY. E. R. SALEEBY, J. A. M. A. **85**:1131 (Oct. 10) 1925.

The author examined the seminal vesicles from twenty-eight definitely syphilitic cases. In none were definite syphilitic changes found, though in two the blood vessels in the serosa showed perivascular round cell infiltration. Sections stained for *Spirochaeta pallida* were negative. The findings indicate that syphilis of the seminal vesicles is rare.

A CASE OF MARKED SPLENOMEGALY AND LYMPHOCYTOSIS ASSOCIATED WITH SYPHILIS. A. ELKIN and H. M. BOWCOCK, J. A. M. A. **85**:1135 (Oct. 10) 1925.

In this unusual case, there was marked splenomegaly and a high lymphocyte count improving with roentgen-ray therapy, and later, reappearance of splenomegaly, secondary anemia, leukopenia, enlarged liver and jaundice, and the discovery of a positive Wassermann reaction, with rapid and marked improvement with antisyphilitic treatment.

A CASE OF CYSTICERCUS OF THE SKIN. Z. B. COBLENTZ, J. A. M. A. **85**:1136 (Oct. 10) 1925.

The patient had a number of small movable subcutaneous nodules. One, which was removed for examination, revealed the head of a tapeworm. The patient previously had experienced an attack of syncope with partial paralysis that suggested the lodgement of a cysticercus in the fourth ventricle.

CHEMICAL ANALYSIS OF SWEAT. R. E. BARNEY, J. A. M. A. **85**:1373 (Oct. 31) 1925.

By placing his subject between rubber sheets and using heat, forcing fluids and giving pilocarpin, Barney obtained large quantities of sweat, up to 690 c.c. in an hour and a half. Before analysis, it was filtered. It was analyzed for total solids, urea nitrogen, and nonprotein nitrogen. Fourteen normal subjects were first investigated so as to establish normal standards. The normal average nonprotein nitrogen per 100 c.c. of sweat is more than twice that in the blood and the urea nitrogen slightly higher. In six cases of senile eczema there was an appreciable diminution in the output of nitrogenous elements of the sweat. This may have some bearing on the cause of this disorder. Six cases of psoriasis showed no appreciable change from the normal.

MICHAEL, Houston, Texas.

RESULTS OF SURGICAL TREATMENT OF CANCER. G. W. CRILE, New York State J. M. **25**:1019, 1925.

This is a concise report of the results of the various methods of treating cancer. Radium therapy is recommended for all cases of carcinoma of the skin, with the exception of the pigmented mole, which should be excised. No mention is made of any distinction between basal cell and prickle cell epithelioma of the skin. The electric cautery is recommended for early cancer of the mucous membrane of the gums or cheeks, which do not require excision of the lymph nodes. In later cases, complete removal of the associated lymphatic system is required. Radium therapy is the preferred method in early cases of cancer of the lip, while later cases require V shaped incision. Again, no distinction is made between basal cell and prickle cell carcinoma. For early

cancer of the tongue, electric coagulation or cauterization is preferred, as being as effective as radium and less painful; complete removal of the associated lymph nodes is required. The rest of the article deals with cancer in the deeper structure of the body.

ROENTGEN-RAY IN CANCER THERAPY. B. F. SCHREINER, New York State J. M. **25**:1025, 1925.

This is a discussion of the value and the applicability of unfiltered roentgen rays, low voltage roentgen rays with moderate filtration and high voltage roentgen rays with heavy filtration. Unfiltered roentgen rays are recommended for all superficial lesions, such as basal cell epithelioma, pigmented nevi and epithelioma of the lip and penis. Basal cell epithelioma can be healed in 95 per cent. of the cases by means of a full erythema dose of unfiltered roentgen rays. One or two treatments usually suffice. In the treatment of prickle cell epithelioma, such as occurs on the lip, skin and penis, the author reports a primary healing in 75 per cent. of the primary cases, of which 50 per cent. are apparently permanent. It is often necessary to use from two to three times the erythema dose. The rest of the article is devoted to deep therapy. It calls attention to the danger of toxemia from massive dosage and to facts which prove that the curative power of the rays is due, in part at least, to an effect on the normal tissue.

WILLIAMS, New York.

CHRONIC HEMORRHAGIC PURPURA CURED BY SPLENECTOMY. A. CHAUFFARD and PIERRE DUVAL, Presse méd. **33**:961 (July 18) 1925.

The authors cite an interesting case of chronic hemorrhagic purpura, which had existed at varying intervals since the patient had been 5 years old. At the age of 16, after numerous hemorrhages, a splenectomy was performed, and the patient was cured.

Splenectomy is also curative for congenital icterus. It is, of course, a dangerous operation, and should be performed only when absolutely necessary to save a life. However, the operative results are improving.

THE VARIATIONS OF THE FREQUENCY OF MIXED CHANCRE. HUDELO and RABUT, Presse méd. **33**:1143 (Aug. 29) 1925.

The authors are of the opinion that cases of mixed chancre are decreasing. They state that cases of soft chancre have increased around Paris since 1919; that the number of cases of syphilitic chancre has decreased between the years 1919 and 1924. They believe that the decrease in cases of mixed chancre is due to the decrease in the syphilitic chancre. They give statistics to bear out their conclusions.

MCCAFFERTY, New York.

CONTRIBUTION TO OUR KNOWLEDGE OF SYRINGOMA. OSKAR KRESS, Dermat. Wchnschr. **80**:1 (Jan. 3) 1925.

The author describes in detail a case of syringoma of generalized distribution observed over a long period of years and studied with repeated histologic examinations. He calls special attention to two clinical facts: there was intense itching, worse at night when the patient's body was warmed up in bed; lesions almost imperceptible in the early part of the day became dusky red, elevated and very noticeable during the afternoon. The sweat secretion

was normal and the injection of pilocarpin caused no changes in the appearance of the lesions and had no effect on the secretion of sweat. The histologic studies confirmed findings in former cases. In only one instance was any connection found between the cysts and cell strands with the sweat glands and ducts. There was no relation between the cyst and cell strands and the blood vessels, no red blood cells being found in the former. Normal sweat glands were present even in the regions where the syringoma lesions were most profuse.

A Case of Brown Chromidrosis. A. M. Schoch, Dermat. Wchnschr. **80**:12 (Jan. 3) 1925.

The author reports a case of brown chromidrosis involving particularly the nails, the hands and trunk, and accompanied by severe constitutional disturbances. As the patient's sweat gave negative cultures for chromogenic bacteria, and since all drugs could be ruled out, the case was explained on an idiopathic basis.

One Hundred and Forty Cases of Alopecia Areata. Arthur Jordan, Dermat. Wchnschr. **80**:85 (Jan. 17) 1925.

After a careful analysis of his cases extending over a period of ten years, the author comes to the following conclusions: (1) There is no evidence to support the parasitic theory. (2) Syphilis is only a coincidence in alopecia. (3) Injuries, trophoneurotic disturbances and changes in the sympathetic nervous system are responsible for the large majority of the cases.

The Problem of the Transmission of Hereditary Syphilis. H. Rietsche, Dermat. Wchnschr. **80**:91 (Jan. 17) 1925.

This is an excellent review of this subject and cannot be dealt with fairly in abstract form. It should be read in the original to be appreciated properly.

An Unusual Dermatosis Occurring in Polycythaemia Rubra. Erwin Pick and Carl Katznelson, Dermat. Wchnschr. **80**:159 (Jan. 31) 1925.

The authors report a peculiar universal inflammatory involvement of the skin occurring in a man with polycythemia rubra. This patient also showed an extremely high grade type of vasomotor irritability. The eruption, accompanied by intense itching, began as an urticarial, rosy red papule the size of a split pea, which in the course of further development took on a brown color and, at first being surmounted with a vesicle and then a pustule, formed a crust. Healing was followed by pigmentation and a superficial soft scar. Although the distribution was universal, the face, neck and shoulders were most involved. All forms of local therapy were used for this eruption, but to no avail, until the long and flat bones were given heavy doses of roentgen ray. Immediately after this the skin eruption and the polycythemia cleared up. The success of roentgen-ray treatment after every local treatment had failed caused the authors to regard the etiology of the skin eruption as that of polycythemia, and they suggest the name acne urticata polycythemia for the former.

Surgical Treatment of Persistent Pruritus Ani; Treatment of Severe Chronic Circumscribed Eczema. Frankenthal, Dermat. Wchnschr. **80**:163 (Jan. 31) 1925.

Frankenthal believes that surgical intervention in the form of excision of the involved area of the skin and resection of the nerve (N. perineus) is the

most successful treatment of resistant pruritus ani. In pruritus vulvae a resection of the posterior labial nerve is indicated. Similar surgical procedures should be undertaken in patchy, indurated eczema accompanied by intense itching and resistant to all other forms of treatment for a period of several years.

McCarthy, Washington, D. C.

DERMATOLOGIC ABSTRACTS

The Journal of the American Medical Association

Efficiency and Inefficiency of Certain Skin Antiseptics. M. B. Tinker and H. B. Sutton, Ann. Surg. **82**:640 (Oct.) 1925.

Tinker tested the efficiency as skin antiseptics of iodin, picric acid, Harrington's solution, mercurochrome, acriflavine and acriviolet. Acriflavine, 5 per cent. in 50 per cent. alcohol, proved most efficient. Next came acriviolet, 2 per cent. in 50 per cent. alcohol; then 5 per cent. alcoholic iodin; acriviolet, 10 per cent. aqueous, and Harrington's solution. Picric acid, 5 per cent., in 95 per cent. alcohol, and mercurochrome, 5 per cent. in 50 per cent. alcohol, were the least effective.

Germicidal Efficiency of Coconut and Linseed Oil Soaps. F. W. Tilley and K. M. Schaffer, J. Infect. Dis. **37**:359 (Oct.) 1925.

Tilley and Schaffer found that coconut oil soap has a greater bactericidal efficiency than linseed oil soap against *B. typhosus, B. pyocyaneus* and *Staphylococcus aureus,* both in the absence of added organic matter and in the presence of 25 per cent. of the horse blood serum. The bactericidal efficiency of coconut oil soap is increased by the addition of a slight excess of alkali and by the addition of sodium chlorid. The practical value of the action of sodium chlorid is, however, greatly diminished by the undesirable physical changes produced and by failure to maintain the increased efficiency in the presence of blood serum. Although its low efficiency against *Staphylococcus aureus* and *B. tuberculosis* limits the value of coconut oil soap as a disinfectant, it seems likely that it can be used to advantage against other organisms. It is suggested that if a nonpoisonous antiseptic in the form of soap is desired, this need may be very well met by the use of pure coconut oil soap, which combines some germicidal activity with the detergent properties of soap. The bactericidal efficiency of cresol soap mixtures is greatly affected by the proportions of soap and cresol used. Mixtures containing approximately half as much soap as cresol showed maximum efficiency. An increase in the proportion of soap causes a reduction in efficiency. Saponified cresol solutions containing coconut oil soap have a greater germicidal efficiency than those containing linseed oil soap, both in the absence of added organic matter and in the presence of 25 per cent. blood serum. The difference in efficiency is most marked when the cresylic acid used contains a large proportion of high boiling phenols. The addition of sodium hydroxid in fairly large excess over that needed to saponify the oil does not diminish the bactericidal efficiency of saponified cresol solutions. The addition of sodium chlorid to diluted saponified cresol solutions containing coconut oil soaps increases bactericidal efficiency without causing

such undesirable physical changes as occur with the soaps alone. The increased efficiency is fairly well maintained in the presence of 25 per cent. blood serum. It was found that 21 per cent. coconut oil (or the chemically equivalent amount of its fatty acids) as soap can satisfactorily take the place of 28 per cent. linseed oil as soap for holding in solution the 50 per cent. cresylic acid contained in saponified cresol solutions.

PSORIASIS TREATED ROENTGENOLOGICALLY. J. O. PARKER, J. Radiol. **6**:389 (Oct.) 1925.

Parker asserts that complete cure in 85 per cent. of cases of psoriasis can be had by the employment of the roentgen ray alone, provided the patient acquiesces to our demands. A general stimulation of the organic functions during and following the course of treatments may be expected. Succeeding generations will not suffer from induced malformations, when guarded dosage is employed.

TREATMENT OF SYPHILITIC EXPECTANT MOTHER. E. R. HALL, Southern M. J. **18**:757 (Oct.) 1925.

Hall believes that by educating and treating the expectant syphilitic mother more can be accomplished toward eradicating syphilis than by any other means. Wassermann examinations should be made on all pregnant women. Next to a Wassermann test, the history of a previous abortion, miscarriage, stillbirth or living syphilitic child is the most valuable sign. In only about 18 per cent. of cases detection is aided by previous personal history or clinical signs. Arsphenamin and neo-arsphenamin are the drugs of choice. Every syphilitic expectant mother should be treated with arsphenamin or neo-arsphenamin. Treatment is most effective when given early in pregnancy. All patients in this series having a syphilitic child started treatment after the sixth month of pregnancy. A small amount of treatment early in pregnancy will often result in a healthy child. The same amount given before conception will in most cases result in a syphilitic child.

DEATH FROM HEMORRHAGE IN SCARLET FEVER. A. CALDER, Brit. M. J. **2**:566 (Sept. 26) 1925.

The hemorrhage in Calder's case occurred on the thirty-fourth day of the disease, from the external jugular, which ruptured and the child died. Incident to a very severe sore throat a great deal of sloughing resulted, exposing the vein and the carotid artery to view.

VALUE OF COMBINED TREATMENT OF SYPHILIS. BERNARD, Bruxelles méd. **5**:1450 (Oct. 4) 1925.

Bernard examined the cerebrospinal fluid of 200 syphilitics who had responded negatively to the Wassermann tests of the blood for periods ranging from six months to several years. The cerebrospinal fluid was normal in eighty-two of the patients, and pathologic in the remaining 118. About 72 per cent. of the subjects with a normal cerebrospinal fluid had been treated with arsenicals and mercury; and only 17 per cent. with arsenicals alone. About 14 per cent. with a pathologic cerebrospinal fluid had been treated with arsenicals and mercury; 63 per cent. with arsenicals alone. The simultaneous use of both drugs seemed to be more effectual than alternating the mercury and arsenical series.

TREATMENT OF FURUNCLES OF THE FACE. R. MORIAN, Deutsch. Ztschr. f. Chir. **193**:45 (Sept.) 1925.

Morian asserts that the malignant furuncle with sepsis from the start is doomed, whatever treatment is applied. In the 103 cases of furuncles on the face in Payr's service, 1913-1924, 10.7 per cent. were of this fatal type. The others all healed under conservative measures, salve, protecting bandage and fluid food. The results were equally good in former years under incision, but no better, and the disfigurement is less.

HONGKONG FOOT. G. N. FRAZIER, China M. A. J. **39**:705 (Aug.) 1925.

Hongkong foot is an infectious disease of mycotic origin found in North, Central and South China. In Peking, during the summer months, it is commonly seen and occurs with equal frequency among Chinese and foreigners. The initial symptom is a persistent, intolerable itching of the hollow of the sole, or of the toes and the interdigital spaces. This symptom does not disappear with the development of cutaneous lesions. These manifest themselves in one of two forms, or more frequently in a combination of the two. The first and more common consists of small, deep-seated, epidermal vesicles, which contain a clear serum. These are usually situated on the inner margin of the hollow of the sole; on or between the toes, especially the third, fourth and fifth digits at their proximal extremities; and on the plantar surface near the toes. The great and second toes are conspicuously avoided by the infection. With evolution of the vesicular lesions their coverings become thinner and usually rupture, discharging a sticky, serous fluid, after which rapid desiccation and exfoliation ensues. New lesions continue to form throughout the active period of the disease. With the onset of cold weather the process has a tendency to subside. Not uncommonly through traumatism and lack of care, secondary pyogenic infection complicates the picture. The vesicular type is found on the hands, concomitant with or independent of, similar lesions of the feet. Hongkong foot is not peculiar to China. Under the name of eczematoid dermatitis of the extremities, ringworm of the hands and feet, Dhobie itch of the toes, epidermophytosis, etc., the disease has been described in Europe and America (where it is quite common) and in tropical countries.

Society Transactions

PITTSBURGH DERMATOLOGICAL SOCIETY

Regular Meeting, Oct. 15, 1925

LESTER HOLLANDER, M.D., *President*

KERATOSIS FOLLICULARIS — DARIER'S DISEASE. Presented by DR. PHILLIPS.

F. H., a man, aged 55, a blacksmith, presented a skin eruption which had existed since he was 7 years old, gradually growing worse. Itching and soreness were present, and the odor was offensive. Practically every part of the body showed pigmented macules and papules; however, the only areas showing papillomatous, vegetating, tumor-like growths were the chest, pubic region and shins. These were covered with vegetating masses from which pus exuded on slight pressure. A few areas had broken down to form ulcers, the largest of which was about the size of a half dollar. Many other areas were covered with a brown greasy crust, especially the scalp, ears, axillae, groins and back. The crust over the scalp was not unlike that found in severe cases of seborrhea, except that it was more firmly adherent. The patient's father and one of his sons suffered with a similar condition. Two brothers, one sister and the other son were free from skin lesions.

DISCUSSION

DR. HOLLANDER: This unusual case illustrates better than any other case of this type the follicular papules with the concomitant keratinization. About five years ago I treated a young woman with roentgen-ray irradiation, whose forehead, neck and extremities were affected. She improved considerably.

ANGIOMA. Presented by DR. CRAWFORD.

(This case was presented at the annual meeting of the Pittsburgh Dermatological Society, April 6, 1925.)

L. S., aged 2, had had a large pulsating subcutaneous angioma of the right cheek. The mass had almost completely disappeared with filtered sub-erythema roentgen-ray irradiation, without injury to the epidermis.

DISCUSSION

DR. GUY: I should be interested to learn whether any degenerative skin changes appeared during the next few years. Were the underlying glandular structures apparently affected?

DR. CRAWFORD: I have been anxious to try filtered roentgen rays in the treatment of this case, and the result has been satisfactory. I believe that filtered roentgen ray and gamma radiation of radium are similar and that a good result can be had with either. I avoided the parotid gland area in the treatment of the case. I did not note any change in the tonsils, which were not enlarged before treatment.

SYPHILIS. Presented by DR. HOLLANDER and DR. GOLDMAN.

Mr. H. C. P., aged 40, an American, presented on the left arm and over the left side of the lower portion of the chest, rounded, vaccinia-like scarring. There were a great number of these scars. They were nonconfluent, and the individual scar was round, covered by a thinned out white skin with a suggestion of yellowish pigmentation about it. The distribution gave a suggestion of herpes zoster. This condition began in 1918 with a small pustule formation which followed an injection of vaccine, and continued over a period of two years. It cleared up after administration of arsphenamin. The patient's blood Wassermann test was 4 plus.

The patient's wife presented a unilateral eruption on the right wrist composed of three brownish nodules separated by a slight cigaret-paper-like scarring. The patient had been married fifteen years; his wife had not been pregnant. The blood Wassermann test was weakly positive.

DISCUSSION

DR. GUY: I am inclined to accept syphilis as responsible for the underlying cause, although the appearance of the arm suggests the possibility of narcotic addiction. I feel that in view of the history and the fact that the patient's wife presented a tertiary syphilid on the forearm, together with the appearance of atrophic macules, that the case should be reported as macular atrophy due to syphilis.

DR. CRAWFORD: I consider the lesions of artificial origin following injections of morphin. The lesions are grouped on the left upper arm and over the epigastrium in areas offering easy access for this purpose. The linear group of small, round, atrophic areas on his back, which followed electrical burns, would lead one to believe that puncturing trauma was followed by atrophy in the skin.

DR. HOLLANDER: I presented the husband and wife for the purpose of impressing the necessity of examination of husband and wife and children, if there are any, when a diagnosis of syphilis is made. The wife was the first one to appear for examination, and she presented a typical circinate nodular tertiary syphilitic lesion at the wrist. Her blood Wassermann reaction was weakly positive, but her luetin was markedly so. When the husband appeared for examination, these peculiar scars, the remnants of what I consider small individual gummas, were discerned. In accepting these as scars from syphilitic lesions, the history is of the greatest importance, for the patient states that over the period of two years these small nodular lesions were forming, which would break down in the center. After the administration of five doses of arsphenamin the condition disappeared.

SCLERODERMA (CIRCUMSCRIBED). Presented by DR. HOLLANDER.

Miss V. S., aged 18, American, presented multiple, elongated, irregular, ribbon shaped plaques showing depression and pigmentation and arranged along the distribution of the cutaneous nerves of the thigh and arms. The lesions were symmetrical. The patient was shown to demonstrate effect of pituitary feeding and roentgen-ray treatment.

The case is of interest only because with the administration of roentgen ray and pituitary substance anteriorly, there seems to be a gradual softening of the plaques of scleroderma. I want to warn against the use of thyroid gland extracts, except when the patient can be observed closely, as the con-

tinued use of it, without supervision, may lead to a thyroid toxicosis. I had an opportunity to see a case of this type with Dr. Guy in which, on the recommendation of an excellent dermatologist, the patient was taking thyroid gland extract. She, however, was not under his supervision, and when seen by us showed grave symptoms of thyroid toxicosis.

DERMATITIS VENENATA CHRONIC. Presented by DR. HOLLANDER.

Mr. F. C., aged 53, American, presented a vesicular eruption on the forehead, arms and about the buttocks. The condition was of seven months' duration and had peculiar configuration on the forehead. The case was presented on account of the persistency of the lesions and the significance of the history. The salient features of it are that within one week after the appearance of the lesions the patient consulted a dermatologist, who, diagnosing the condition as impetigo contagiosa, asked the patient to remain at home, and with the application apparently of calamin lotion and staying at home, the lesions cleared up. The lesions recurred immediately after the patient went back to his work, that of an engineer handling blue prints. The condition was always worse in the evening after the patient returned from work. On another occasion the patient was improved while he was on his vacation and away from his work. All this suggests the possibility of irritation from substances that the patient came in contact with during his work.

The case was presented because it was unusual. There were a number of scattered vesicular patches on the extremities and on the body, and a marked area of involvement on the forehead and face. Vesicular, well defined with a raised border, intense burning, which was relieved only when serum exuded, was the clinical picture presented. The patient lived next to a vacant lot, which was full of weeds. Dr. Hollander said that he would report on the progress of this case at the next meeting, and that he would institute treatment for chronic dermatitis venenata.

FRED M. JACOB, M.D., Secretary.

Book Review

RADIUM THERAPY. By FRANK EDWARD SIMPSON. Pp. 391, with 166 original engravings. St. Louis: C. V. Mosby Company, 1922.

A text on radium therapy by one whose clinical experience with its use has been so extensive as that of Dr. Simpson could be expected to furnish a useful and reliable addition to the literature of this field, and perusal of the book bears out this belief.

The text, although not large, offers a comprehensive review of the modern science of radiology, including, as it does, a consideration of every phase of the subject, from the origin and chemical nature of radium to its therapeutic application. The bibliography, which contains fifty-nine pages, attests the full investigation which underlies the book.

The first ten chapters, comprising eighty-six pages, are concerned with the technical and theoretical phases of the subject, covering radioactive substances in general; the origin and chemical nature of radium, radium emanation and radioactive deposit, with the technic of preparation of the former for its therapeutic use; absorption and filtration of rays; physical and chemical effects of radium rays and their biologic effects. Included here is a rather long and largely technical chapter covering the author's experimental work on the absorption of gamma rays. On the whole, however, considering the highly specialized nature of the subject, this portion of the book is written in such a style that the greater part of it can be easily understood by the physician who has no special knowledge of the physics of radium.

The next four chapters, dealing with radium reactions, therapeutic apparatus, dosage and the technic of radiation present the general considerations of radium therapy in adequate fashion, although the formulas used in determining dosages are so complicated that the average reader will find himself beyond his depth.

Following this is a discussion of the use of radium or its emanations in the special fields of medicine, with a full statement of the form of the agent best suited for the special condition; type and thickness of screens to be employed, and mode and time of application for each disease in which its use is advised. In the chapters dealing with its employment in general surgery and gynecology the author displays a conservative and open-minded attitude which is likewise evidenced in the chapters on radium in dermatology, diseases of the eye, ear, nose and throat, diseases of the ductless glands, and internal medicine. The instructions for its employment in the various conditions are so clearly and simply stated and warnings against overdosage so emphasized, that the casual user of radium finds this an excellent guide for his efforts. In many instances, however, the quantity of radium recommended is so large that not many physicians will have the suggested amount available.

The closing chapter deals with the professional injuries due to radium.

The book is liberally illustrated with engravings which include various charts, depiction of apparatus and a number of clinical photographs showing the condition before and after treatment. Most of the latter illustrate gratifying results, but others are included, which, like the text, do not hesitate to record the limitations of the agent employed.

Simpson's text can be highly recommended as an able and modern exposition of the subject of radiology, of value to physicians generally as well as to those actively occupied with radium therapy.

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